1986

The Relation Among Stressful Life-Events, Affective Responses, and Headaches (Migraine, Muscle-Contraction).

Craig Douglas Waggoner
Louisiana State University and Agricultural & Mechanical College

Waggoner, Craig Douglas

THE RELATION AMONG STRESSFUL LIFE-EVENTS, AFFECTIVE RESPONSES, AND HEADACHES

The Louisiana State University and Agricultural and Mechanical Col. PH.D. 1986

PLEASE NOTE:

In all cases this material has been filmed in the best possible way from the available copy. Problems encountered with this document have been identified here with a check mark __✓__.

1. Glossy photographs or pages _____
2. Colored illustrations, paper or print _____
3. Photographs with dark background _____
4. Illustrations are poor copy _____
5. Pages with black marks, not original copy _____
6. Print shows through as there is text on both sides of page _____
7. Indistinct, broken or small print on several pages ✓
8. Print exceeds margin requirements _____
9. Tightly bound copy with print lost in spine _____
10. Computer printout pages with indistinct print _____
11. Page(s) _____ lacking when material received, and not available from school or author.
12. Page(s) _____ seem to be missing in numbering only as text follows.
13. Two pages numbered _____. Text follows.
14. Curling and wrinkled pages _____
15. Dissertation contains pages with print at a slant, filmed as received _____
16. Other _____

University
Microfilms
International

THE RELATION AMONG STRESSFUL LIFE-EVENTS, AFFECTIVE RESPONSES, AND HEADACHES

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the degree of
Doctor of Philosophy

in

The Department of Psychology

by

Craig D. Waggoner
B.S., Ohio State University, 1978
M.A., Bradley University, 1980

May 1986

Acknowledgements

I wish to extend my greatest appreciation to my major professor, chairman of my dissertation committee, and good friend, Dr. Phillip J. Brantley. He gave freely of his time, knowledge, and thoughful advice, challenged me and stimulated my best efforts, and provided me with encouragement throughout this project. I hold the highest regard for him, both personally and professionally, and greatly value his friendship.

I also extend many thanks to Dr. Frank M. Gresham and Dr. Ray Buss for their guidance through the complex statistical treatment of the data accumulated in this project and for providing their thoughtful editorial comments and valued feedback as members of my committee.

Much appreciation goes to Dr. William F. Waters and Dr. Arthur Riopelle. They were active and supportive committee members who stimulated my thinking and helped me untangle the complex theoretical concepts that were addressed in this project.

Special thanks are given to my colleague and friend, Anthony Goreczny, who spent numerous hours assisting with the collection, scoring, and analysis of the data. He was a key member of the research team involved with this project.

Special thanks are also extended to two other colleagues, David P. McAnulty and Neil B. Rappaport, who collaborated with me on this project from the very

beginning and helped see it through to its completion.

I also wish to extend my appreciation to my classmates and friends, all of whom generated an atmosphere of creativity and comaradery that made the entire process more pleasant.

My warmest thanks go to my parents, Nan and Ray, who have been supportive of me throughout my academic career. Only with the relentless love, understanding, and encouragement, that only parents can give, was reaching this goal possible.

Finally, I want to thank a very special person, Elizabeth Hunt, who was a never ending source of love, support, and encouragement during the long hours of writing and revising this manuscript.

Table of Contents

List of Tables

List of Figures

List of Appendices

Abstract

Stress is frequently reported as a major contributing factor in both migraine and muscle-contraction headaches. The general conclusions of studies investigating the stress-headache relations are that headache sufferers experience a significant amount of stress in their lives, that stress can precipitate headache attacks, and that headache subjects tend to overreact to stress. The laboratory method and scales measuring major life-events have typically been used in studying stress but have several limitations, particularly for studying exacerbations and remissions of symptoms. Increasing the comprehensiveness of stress measurement, beyond the traditional life-events approach, by assessing stress and affective states on a daily basis was recommended by the reviewed literature. The present study examined the amount of stress present in the lives of headache sufferers, whether or not headache sufferers overreact to stressful life-events, the relative importance of major and minor stressful life-events for predicting headache activity, and the role that affective states play in different headache disorders.

Migraine, mixed, and muscle-contraction headache sufferers and control subjects recorded their headache activity, completed the Daily Stress Inventory, and completed the MAACL on 28 consecutive days. Subjects also completed the Life Experiences Survey. There was no

evidence that headache subjects, as compared to controls, experienced a greater amount of life stress or emotionally overreact to stress. Headache subjects, regardless of diagnosis, were more depressed and anxious on headache days as compared to pre-headache days. In addition, the headache groups displayed different patterns of changes in affective states as their headaches approached. Daily stress scores significantly predicted headache activity and improved the predictions made by major life-events scores. The findings advocate using daily measures of stress and affect for studying the stress-headache relations. Implications for stress theories, future research, and treatment, as they pertain to headaches, are discussed.

The Relation Among Stressful Life-Events, Affective Responses and Headaches

An estimated 42 million Americans suffer from headaches, almost 12 million of which are some form of migraine (National Migraine Foundation, cited in Adams, Feuerstein & Fowler, 1980). The high prevelance of this problem is not, however, limited to the United States. European surveys have provided estimates that 14 to 70% of individuals in their samples suffered from some form of headache (Leviton, 1978; Waters & O'Connor, 1975; Ziegler, 1978), the most common types being migraine, muscle-contraction, and combined migraine and muscle-contraction headaches. Headaches are also among the ten most common presenting symptoms seen in outpatient medical settings (Bain, 1967; Leviton, 1978). One survey, conducted in the United States, found headaches to be one of the 14 most commonly presented symptoms in outpatient medical clinics, accounting for over 12.3 million office visits during 12 months (DeLozier & Gagnon, 1975).

In 1962, the Ad Hoc Committee on Classification of Headache delineated 15 different categories of headache. Large scale surveys have found that, of these 15 headache types, muscle-contraction, migraine, and combined headaches are the most commonly reported. The Ad hoc Committee (1962) stated that none of these types of headaches are the result of cranial structural

abnormalities.

Approximately 80% of all headaches are believed to be of the muscle-contraction (also called "tension") type (Ostfeld, 1962; Waters & O'Connor, 1971). These headaches are usually bilateral (but can be unilateral), with the pain generally located in the occipital regions but also radiating to the frontal, parietal, and temporal regions (Diamond & Dalessio, 1978; Friedman, 1979). The pain is typically described as a steady, nonpulsating ache or sensation of tightness or pressure; often like a "band" about the head; that may vary greatly in intensity, frequency, and duration (Ad Hoc Committee, 1962; Adams et al., 1980; Appenzeller, Feldman & Friedman, 1979; Diamond & Dalessio, 1978).

Like muscle-contraction headaches, migraine headaches can also vary greatly in intensity, frequency, and duration (Ad Hoc Committee, 1962). A migraine attack is usually characterized by a pulsating pain that has a sudden, unilateral onset (but may also be bilateral); is frequently accompanied by anorexia, nausea and, sometimes, vomiting; is often accompanied by increased sensitivity to light, sound, and some odors; and sometimes accompanied by diarrhea or constipation (Ad Hoc Committee, 1962; Diamond & Dalessio, 1978; Williamson, 1981). The head pain, which is typically located in the temporal, orbital, supraorbital, or occipital regions usually lasts from 6 to 8 hours but can last up to several days (Adams et al.,

1980; Diamond & Dalessio, 1978). The Ad Hoc Committee (1962) also made a distinction between "classic" and "common" migraine headaches. The most significant characteristic that differentiates these two forms of migraine is that the former are preceded by a prodromal phase (10-30 minutes prior to the experience of pain) during which visual disturbances (e.g., scotomata, teichopsia, flashes of light), auditory distubances, and paresthesias are reported (Diamond & Dalessio, 1978). Common migraine is the most frequently reported type of migraine comprising approximately 85 to 90% of all migraine cases (Friedman, 1963, 1978; Kudrow, 1978).

The descriptions of muscle-contraction and migraine headaches, presented above, represent the typical clinical pictures. In reality, the distinction between these two types of headaches is less clear than these descriptions indicate. In fact, there is considerable overlap of symptoms between them (Appenzeller et al., 1979; Friedman, von Storch & Merritt, 1954; Ziegler, 1979). Acknowledging the fact that many individuals experience headaches presenting with both muscle-contraction and migraine symptoms, the Ad Hoc Committee (1962) established the category of combination (or mixed) headache. Most often this diagnosis is given to individuals who report distinct episodes of both migraine and muscle-contraction headaches. Recently, however, Saper (1983) has proposed that subgroups of individuals within this category may

experience both muscle-contraction and migraine symptoms during the same headache. The problems in finding clearly distinct headache categories have led some to view headache symptoms as lying on a continuum with purely muscle-contraction and purely migraine symptoms at opposite ends (Bakal & Kaganov, 1979; Drummond & Lance, 1984; Philips, 1978).

Over the last three decades, researchers have given increased attention to the investigation of headaches, the result of which has produced volumes of articles and books. Much of this research has focused on the assessment of not only the characteristics of these headaches but also the physiological, psychological, and psychophysiological mechanisms that may be involved. The goal of these efforts has been to further differentiate factors involved in the different headache types and also to provide some theoretical bases for the treatment of these ubiquitous disorders. The present manuscript reviews the major biological, psychological, and psychophysiological accounts of migraine and muscle-contraction headaches. In addition, the available evidence pertaining to the roles that environmental and emotional stress play in the production of these headaches, as implicated by these theories, is critically reviewed. Further, the problems and limitations of the existing research investigating the relation between stress and headaches are discussed. Finally, a study is

proposed to correct many of the existing problems apparent in this body of literature in the hopes of not only clarifying theoretical issues but also providing additional information pertinent to the treatment of migraine, muscle-contraction, and mixed headache.

Biological Theories for Migraine and Muscle-Contraction Headaches

Migraine Headaches

Numerous, though not necessarily competing, hypotheses have been suggested for the etiology of migraine headaches. Researchers have sought evidence for the etiology of migraines through investigations of vasomotor functioning, biochemical factors, dietary factors, hormonal factors, electroencephalographic abnormalities, and genetic contributions.

Vasomotor functioning. Changes in cerebral blood flow have been well documented as occurring during migraine attacks (Sakai & Meyer, 1978; Skinhoj, 1973; Tunis & Wolff, 1952). O'Brien (1971) found a 23% reduction in cerebral blood flow during migraine prodromata using the 133 Xenon inhalation method. Research reported by Wolff (1963) and later, Dalessio (1978), delineated a distinct progression of vasomotor changes just before, during, and following a migraine attack. Their research showed that just prior to the painful attack there is vasoconstriction of the cranial arterial beds, followed by vasodilation and the subjective

experience of pain during the attack, and eventual return to normal vasomotor tone of the cranial vasculature following the headache attack. Such observations have led to the postulations of the presence of an overactive vasoconstrictor mechanism (Appenzeller, 1969) or a more general problem with autonomic stability (Tunis & Wolff, 1953) that could produce the abnormal vasomotor activity.

Biochemical Factors. Biochemical theories have been based on the observations that the major symptoms of a migraine headache suggest that there is an increase in parasympathetic tonus, a decrease in sympathetic tonus, or a combination of both occurring during a headache attack (Sacks, 1970). Various vasoactive humoral agents have been implicated in the pathogenesis of migraines based on observed excesses or deficiencies of them during and following attacks. Specifically, histamine (Horton, 1956), acetylcholine (Kunkle, 1959), and serotonin (Anthony, Hinterberger & Lance, 1967; Sicuteri, 1959) have received the most attention. For example, Anthony and associates (1967) estimated that serotonin levels fall to 60% of pre-headache levels during the attack itself. In addition, they found injections of serotonin improved headaches (see Appenzeller, 1976; Botney, 1981; and Sacks, 1970 for extensive reviews of this literature). More recently, adenosine triphosphate (Burnstock, 1981), substance P (Moskowitz, Reinhard, Romero, Melamed & Pettibone, 1979), one of the most powerful vasodilating

substances known, and the plasma kinins (Dalessio, 1978) have also been implicated in migraines.

Appenzeller (1969) has suggested that various humoral vasoconstricting agents are present in excessive amounts, at times, which produces the initial excessive cranial vasoconstriction and subsequent prodromata as a result of ischemia. When the concentration of these humoral agents changes again, the vasoconstriction gives way to vasodilation of the cranial arteries. The rapid change in the diameter of the vessels causes the pain associated with migraine headaches according to this theory (Appenzeller, 1969). An alternate theory has been proposed by Diamond and Dalessio (1978). Their "Unified Theory of Migraine" holds that various stimuli and environmental stressors could produce the initial constriction of the extracranial blood vessels. These vessels are innervated by adrenergic fibers that are respondent to catecholamines. This vasoconstriction is thought to be responsible for producing the prodromata in migraines as a result of anoxia and acidosis and, through hemodynamics, to lead to vasodilation of the intracranial blood vessels. The intracranial arteries are not innervated by adrenergic fibers and, thus, not reactive to external stimuli and stressors but, rather, react to sustain the metabolic needs of the brain tissues. According to the Unified Theory, the sustained constriction of the intracranial vessels produces rebound

dilation of the extracranial vasculature which causes the release of vasoactive substances and produces a sterile inflammation, edema, and the throbbing pain characteristic of migraine headache. Botney (1981) hypothesizes that cerebral hypoxia and ischemia (a result of vasoconstriction) results in an inhibition of serotonin synthesis that leads to an increased perception of pain. The theories described above differ mainly in the ordering of the events that are postulated to occur to produce a migraine headache. Whether vasoactive substances produce the initial vasoconstriction, are released subsequent to vasodilation, or their production is inhibited by vasomotor changes remains a point of debate (Willlamson, 1981).

Dietary factors. Considering the strong implications for the role of biochemical agents in migraine, the role of foods, containing vasoactive substances, have also been subject to investigation. The ingestion of alcohol, foods containing tyramine (e.g. some cheeses and chicken livers), sodium nitrite (e.g. found in cured meats), monosodium glutamate (e.g. often found in Chinese foods and other seasonings), and carbohydrates have been found to be associated with the occurrence of migraine headache (Diamond & Dalessio, 1978; Johnson, 1978). Selby and Lance (1960) estimated that the ingestion of certain foods such as chocolates, fried foods, fats, and oranges appeared to elicit migraines in

25% of 339 migraine sufferers. Surprisingly, considering the strong implications for the role of foods in migraines, controlled research is scant in the literature to date and conclusions seem to be drawn mostly from clinical observations.

Hormonal factors. Changes in the patterns of migraine attacks in women have been observed as a result of pregnancy, menstruation, and the use of oral contraceptives (Kudrow, 1978; Somerville, 1972; Waters & O'Connor, 1971). Reports have indicated that up to 80% of women who suffer from migraines cease to have them while pregnant, when intrinsic estrogen levels increase (Blau, 1971; Carroll, 1971; Lance & Anthony, 1966). At least one study, however, found that some women first experienced migraines during pregnancy (Callaghan, 1968). Most often, research indicates that menstual migraine attacks are associated with increased levels of estrogen during the menstrual cycle or by the use of oral contraceptives containing estrogen (Epstein, Hockaday & Hockaday, 1975; Kudrow, 1975; Saper, 1978). Menstrual migraines are most commonly reported during the premenstural period when estrogen levels have reached their peak and are rapidly declining (Somerville, 1972). Somerville (1975) attempted to induce headaches by the administration of estrogen and found that estrogen levels had to be sustained for several days before migraines would occur upon estrogen decline. Further, withdrawal from estrogen use, either as a

contraceptive or in maintenance therapy, has reduced the frequency of headaches in as many as 58-70% of cases (Kudrow, 1975). Although it is generally agreed that estrogen is associated with vascular headache, there are still unresolved issues regarding the specific roles of intrinsic and extrinsic estrogen and the stage during the menstrual cycle that headaches are most likely to occur (Adams et al., 1980).

Electroencephalographic (EEG) abnormalities. EEG abnormalities have been found in approximately 45% of migraine patients, much higher than the 10% incidence in the normal population, as reported in some studies (Rowan, 1974; Selby & Lance, 1960). Early studies reported that many migraine sufferers' EEGs demonstrated excessive fast-wave activity, excessive slow-wave activity, and asymmetrical slow-wave activity (Engel, Ferris & Romano, 1945; Selby & Lance, 1960; Strauss & Selinsky, 1941). Excesses of positive spikes, resembling epileptiform patterns have also been found in the EEGs of migraineurs (Whitehouse, Pappas, Escala & Livingston, 1967). These researchers postulated that an autonomic dysfunction with secondary vascular abnormalities and mediating humoral agents could be the mechanism for migraine headaches. The vasoconstrictive phase of migraine can, presumably, set off seizure activity in one predisposed to seizures but this mechanism is probably rare (Diamond & Dalessio, 1978). As others have pointed out, however, there is not

much evidence to suggest that a specific relation between vascular headaches and EEG abnormalities exists because it has been found to exist equally often in muscle-contraction headache sufferers in at least one study, and they exist in several other behavioral disorders as well (Adams, et al., 1980; Diamond & Dalessio, 1978; Masland, 1978).

Genetic contributions. Studies have found that approximately 50 to 60% of migraineurs have a family history for vascular headaches (Drummond, 1983; Lance & Anthony, 1966; Selby & Lance, 1960). More systematic investigations, however, have found a familial incidence to be in the range of 10 to 26%, much lower than the earlier studies (Lucas, 1977; Waters, 1971). Lucas (1977) studied 1800 pairs of twins from the London Register and found 26% and 13% concordance rates for monozygotic and dizygotic twins respectively. Recently, a genetic hypothesis was supported in one study that found that the onset of a migraine headache problem was earlier if there was a "strong" family history for migraines and that a maternal history appeared to be more important than a paternal history (Steiner, Guha, Capildeo & Rose, 1980). A genetic predisposition for migraine headaches appears to be widely accepted in the current literature if one considers that most researchers include "a history of migraine in one or more first degree relatives" as one diagnostic criterion for inclusion into this group (e.g.

Andrasik, Blanchard, Arena, Teders, & Rodichok, 1982a; Blanchard, Andrasik, Arena, Neff, Jurish, Teders, Saunders, Pallmeyer, Dudek, & Rodichok, 1984). This diagnostic practice is most likely the result of the publication by the Ad Hoc Committee (1962) and early writings by Wolff (1963). The strength of any genetic influence in migraines, however, is still in question (Adams, et al., 1980).

Muscle-Contraction Headaches

While research investigating the biological etiologies of migraine headache has become very specific and molecular in recent years, research investigating the biological etiologies of muscle-contraction headaches has progressed relatively little since the pioneering work of Wolff and his associate (Tunis & Wolff, 1954; Wolff, 1963). Their research implicated sustained, voluntary or involuntary, muscle-contractions of the scalp and neck musculature and, possibly a vascular component in the etiology of this head pain. Unlike migraine headache, dietary and genetic factors are not commonly believed to be as important in the development of muscle-contraction headaches (Diamond & Dalessio, 1978; Drummond, 1983).

Sustained muscle-contraction. The early conclusions drawn by Wolff (Tunis & Wolff, 1954; Wolff, 1963) were based on observations of electromyographic (EMG) activity, of various scalp and neck muscles during headaches, induced by various chemical (e.g. histamine,

saline, ethylmorphine chloride) and physical (e.g. eye prisms, bright light, head screw apparatus) agents. These experiments demonstrated that increases and decreases in pain were related, respectively to increases and decreases in EMG activity. In addition, the Ad Hoc Committee (1962) concurred with Wolff's findings by attributing tension headache to the abnormal contraction of head muscles. Subsequent research has attempted to further investigate the EMG-pain relation with comparisons of EMG activity between headache and nonheadache states and between EMG activity and headache pain reports.

Several studies have found increased head and neck EMG levels during the headache state (Freidman, 1963, Malmo & Smith, 1955; van Boxtel & van der Ven, 1978). More recently, however, studies have not found differences in EMG levels during headache and nonheadache states (e.g. Martin & Matthews, 1978; Phillips, 1977; Pozniak-Patewicz, 1976) nor significant correlations between EMG activity and pain reports (Bakal & Kaganov, 1977; Epstein, Abel, Collins, Parker, & Cincirlpini, 1978; Hart & Cichanski, 1981; Holroyd, Andrasik & Westbrook, 1977). The results of these recent studies lead to the conclusion that muscle-contraction, alone, cannot account for this type of head pain. The findings of Haber, Kuczmierczyk, and Adams (1985) suggest that some of the contradictory results of these studies may be due to researchers falling to distinguish between tension headache sufferers and

psychogenic pain patients. Their findings indicate that the EMG activity of psychogenic pain patients does not correlate with pain while that of tension headache sufferers does.

Dalessio (1978) has proposed that a local pathologic process (undefined) could initiate the cephalic muscle spasm causing continuous stimulation of the gamma efferent neurons. According to Dalessio, the stimulation of these neurons cause the affected muscles spindles to remain contracted and enhance muscle contraction by way of the spinal ventral horn. This theory, while plausible, has not yet been tested.

Vasomotor changes. Wolff's (1963) original conceptualization of the etiology of muscle-contraction headache also included observations of vasomotor changes (Tunis & Wolff, 1954). They found their headache patients to evidence higher pulse-wave amplitudes, than nonheadache controls, during headache-free states and that vasoconstriction of various nutrient arteries occurred with increases in EMG activity. Further support for these observations has been provided by Ostfeld and associates (Ostfeld, 1962; Ostfeld, Reis, & Wolff, 1957) who observed the constriction of conjunctival vessels during these headaches. Others, however have found vasodilations of various other arteries (e.g. in facial capillaries, and frontal and brainstem-cerebelar regions) during muscle-contracation headaches (Onel, Freidman & Grossman,

1961; Raskin & Appenzeller, 1980) thus complicating Wolff's original theory (Williamson, 1981).

Biochemical factors. A survey of the literature of the etiology of muscle-contraction headaches reveals only sparse attention to biochemical factors. This is in marked contrast to the postulated role of biochemical factors thought to be involved in the production of migraine headache (discussed previously). The early work by Ostfeld and his associates (Ostfeld, 1962; Ostfeld et al, 1957) led them to postulate a role for norepinephrine. They suggested that local and circulating norepinephrine increased with feelings of tension and leads to vasoconstriction of the nutrient arteries of relevant head and neck muscles. Pain is increased as a result of ischemia when sustained muscle-contraction occurs. Interestingly, 20 years later, Mathew, Weinman & Largen (1982) have postulated that epinephrine is released in response to acute stress and that norepinephrine is released in response to sustained emotional tension. More recently, amine depletion (Kudrow, 1976) and 5-Hydroxytryptamine (Rolf, Wiele, & Brune, 1981) have also been proposed to play a role in the etiology of these headaches.

Mixed Migraine and Muscle-Contraction Headache

A survey of the headache literature quickly reveals an absence of data on the etiology of mixed headache types. The only conclusions that can be drawn to date

indicate that headaches with mixed symptoms are the result of a combination of the factors believed to be involved in both migraine and muscle contraction headaches (Blanchard et al., 1984). Some authors (e.g. Bakal, 1975; Philips, 1978) have suggested that headache symptoms fall on a continuum and that as initial muscle-contraction headaches become more severe and long standing; they may gain an increasing number of vascular components (Martin, 1983).

Psychological Factors in Headache

The most common description of the migraine headache sufferer's personality offered by early reports is that they tend to be tense, anxious, ambitious, obsessionally perfectionistic, rigid, and have difficulty coping with hostility or anger (Alvarez, 1974; Fromm-Reichmann, 1937; Goodell, 1967; Wolff, 1937; 1963). Similarly, muscle-contraction headache sufferers have been described as rigid, anxious, dependent, compulsive, perfectionistic, and worry-prone and said to display a great deal of denial and depression (Martin, 1966; 1972; Martin, Rome, & Swenson, 1967). As pointed out by many others (e.g. Andrasik et al., 1982a; Harrison, 1975; Philips, 1976) most of these early descriptions are based on clinical impressions and observations rather than objective assessment techniques and are subject to selection biases (i.e., pain complainers). One cannot help but notice the striking similarity in the personality descriptions of these two headache groups. Philips (1976) has noted,

"although two distinctive types of common headache are recognized - migraine and tension - they are not predicted to differ in personality characteristics" (p. 535). The search for the existence of specific personality types for different headaches patterns has continued to the present, and there has been a concerted shift to the measurement of personality characteristics with standardized, objective assessment instruments, such as the MMPI, the Eysenck Personality Inventory (EPI), and the Maudsley Personality Inventory (MPI). The majority of the recent research, using standardized personality assessment instruments, has attempted not only to describe the personality characteristics of headache sufferers but also to compare these characteristics across groups with different headache diagnoses.

Personality Characteristics of Headache Sufferers

Comparisons to normal and other patient populations. In most instances, headache sufferers are found to have mild elevations on the "neurotic" scales (hypochondriasis, depression, and hysteria) of the MMPI, EPI and MPI (Andrasik, Blanchard, Arena, Teders, Teevan, & Rodichok, 1982b; Harrison, 1975; Henryk-Gutt & Rees, 1973; Howarth, 1965; Kudrow & Sutkus, 1979). In addition, Anderson and Franks (1981) found both migraine and muscle-contraction sufferers to exceed normals in anxiety and sympathy seeking. They also provide evidence to support the notion that migraine sufferers are competitive

and perfectionistic and that muscle-contraction headache sufferers are anxious and insecure.

The significance of the psychopathology evidenced in the above studies, however, must be placed into proper perspective. Early, nonobjective, clinical studies claimed that at least 30 to 40% of migraineurs evidenced normal personalities (Dalsgaard-Nielsen, 1965; Selby & Lance, 1960). In addition, the personality scores of migraineurs have been found to be essentially normal in studies using objective measures (e.g. Kudrow & Sutkus, 1979; Sternbach, Dalessio, Kunzel & Bowman, 1980; Weeks, Baskin, Rapoport, Sheftell, & Arrowsmith, 1983). Similarly, Henryk-Gutt and Rees (1973) and Philips (1976) did not find the personality scores of their muscle-contraction headache sufferers to differ from normals. In Philips' (1976) study, none of the headache groups' (migraine, muscle-contraction, and mixed headache) scores on the Eysenck Personality Questionnaire differed from the norms for the general population provided by the authors of the test. In almost every other study, however, at least one headache group has scored significantly different (statistically) from a nonheadache control group (e.g., Anderson & Franks, 1981; Andrasik et al., 1982a; Kudrow & Sutkus, 1979). Although headache sufferers typically score significantly higher than nonheadache normals on personality measures, these elevations rarely reach clinical significance (i.e., T

scores ≥ 70 on MMPI) (Blanchard & Andrasik, 1982; Andrasik et al., 1982a; Sternbach et al., 1980).

Personality scores of headache sufferers have not been found to be significantly different from other medical (nonheadache) populations, who also tend to report physical problems and symptoms of depression and anxiety (e.g., Blanchard & Andrasik, 1982; Watson, 1982). In one study (Howarth, 1965), a muscle-contraction group was found to be significantly less neurotic than patients hospitalized for various other psychosomatic disorders. Another comparison between a combined group of headache sufferers and 50,000 medical patients (from MMPI norms), however, found headache sufferers to score significantly higher on every MMPI clinical scale except scales 4 and 9 (Sternbach, et al., 1980).

In general, the available evidence provided by researchers using objective personality measures agrees with the conclusions of Harrison (1975) that, more often than not, headache sufferers as a group, evidence more psychopathology than normal samples, particularly on scales 1, 2, and 3 of the MMPI. They probably do not, however, differ significantly from other medical patient populations.

Comparisons among headache types. As mentioned earlier, the psychodynamic descriptions of "migraine and muscle-contraction personalities" are highly similar. These descriptions, however, were not based on objective

data amenable to statistical analysis. The possibility of personality differences between migraine and muscle-contraction headache sufferers has been further explored with objective personality measures.

There is some evidence suggesting that migraine and muscle-contraction sufferers present themselves differently on psychological measures. Migraine sufferers have been described as more competitive and perfectionistic and prone to minimize affective expression while muscle-contraction sufferers have been described as more anxious, insecure, hostile, troubled, and confused (Anderson & Franks, 1981; Bihldorf, King, & Parnes, 1971). In addition, headache groups have been found to score significantly different on scales 1, 2, 3, and 7 of the MMPI (Andrasik et al., 1982a; Kudrow & Sutkus, 1979; Sternbach et al., 1980; Weeks et al., 1983). In these studies, the mixed and muscle-contraction groups tended to exhibit more psychopathology than the migraine groups. The post hoc analysis of the Sternbach et al. (1980) study, however, found that only the mixed group scored significantly higher than the migraine group on the MMPI and only on depression scores (scale 2). In contrast, at least two studies found migraine groups to score significantly higher on anxiety, somatization, and neuroticism than muscle-contraction groups (Henryk-Gutt & Rees, 1973; Maxwell, 1966). Further, other studies have not found differences among headache groups (e.g.,

Andrasik et al., 1982b; Philips, 1976). Blanchard and Andrasik (1982) have noted that statistical analyses may not reveal differences among headache groups due to great within-group variability and considerable overlap in the groups' distributions of scores. Much variability in the scores of headache groups has been observed by others (e.g., Bakal, 1975).

More recent studies, employing multivariate statistical procedures, have attempted to discriminate among headache groups on the basis of patterns of scores on various subscales of the MMPI and other measures (e.g., Multiple Affect Adjective Checklist, Beck Depression Inventory). The results of three such attempts generally concur that headache groups can be placed on a continuum according to the degree of psychopathology evidenced. Migraine groups tend to be at the "normal" end, followed by mixed groups and, at the most disturbed end, muscle-contraction groups (Andrasik et al., 1982a; Andrasik et al., 1982b; Kudrow & Sutkus, 1979; Pratt, Williamson, Cohen, Granberry & Jarrel, 1982). In further support of a continuum view, Drummond (1983) observed 600 cases of headaches of varying types and found that as headaches become more frequent, the number of social problems and amount of reported depression also increases. He noted that symptoms of muscular contraction were reported more often by the subjects with more frequently occurring headaches. The results of the above studies

concur that muscle-contraction subjects are generally more anxious, depressed, hostile, and report more psychosomatic symptoms than the other headache subjects. Mixed headache subjects vascillate between being more similar to either the migraine or the muscle-contraction groups on the various measures.

In conclusion, the results of studies comparing headache groups on psychological characteristics remain unclear. On the one hand, the majority of studies have found statistically significant differences between the scores on psychological measures produced by different headache groups with the balance tipped toward the muscle-contraction headache sufferers as evidencing more psychological disturbance. On the other hand, the differences, when found, probably are not of clinical significance and may be more due to recruiting methods, differences in diagnostic criteria, sample sizes used, and the like (Andrasik et al., 1982a; Blanchard & Andrasik, 1982; Harrison, 1975; Philips, 1976). If indeed, the psychological characteristics of headache sufferers are different, then what part do these characteristics play in the production of headaches? The next section describes the various theories related to this question.

The Role of Personality in the Production of Headaches

Psychodynamic formulation. The psychodynamic formulation for headaches relies heavily on the notion of unconscious conflicts. For migraine sufferers, these

conflicts have been hypothesized to be related to hostility, guilt, dependency needs, identifications, and sexual adjustment (Friedman, 1964; Friedman, Van Storch & Merritt, 1954). Similarly, Fromm-Reichman (1937) and Wolff (1937, 1963) considered the inability to adequately deal with hostility as the major conflict evidenced in migraine sufferers. Similarly, for muscle-contraction headache sufferers, unconcious conflicts have been hypothesized to be related to dependency needs, sexuality, and anger control (e.g., Martin, 1966; 1972; Friedman et al., 1954). The inevitable result of these unconscious conflicts is anxiety, which is expressed in the form of muscle-contraction and migraine headaches (Martin, 1983; Philips, 1976). This formulation assumes that individuals with certain personality traits and unconscious conflicts will, especially at times of inordinate stress, develop specific psychosomatic disorders (Alexander, 1950; Dunbar, 1954). In general, these theories have a poor regard for the underlying physiological mechanisms involved in the production of physical disorders such as headaches (Lipowski, 1977). As Harrison (1975) has pointed out, "Only a controlled trial of psychotherapy can demonstrate that personality factors are causally linked to headache" (p. 179). He explained that certain personality characteristics will, under stressful environmental and interpersonal stresses, react with "pernicious" emotions and, if the individual is predisposed to migraines, will

get a migraine headache. One recent study by Sovak, Kunzel, Sternbach and Dalessio (1981) addressed the cause-effect issue with an innovative approach. These researchers gave MMPIs to two migraine groups before and after either a standard drug regimen (propranolol and analgesics) or a thermal biofeedback procedure. Although individuals improved from both treatments, only the group who received the biofeedback procedure showed significant improvement in their MMPI profiles. From these findings, the authors suggest that the psychoneurotic characteristics found in migraine sufferers may be etioloical factors in migraines and not just a product of head pain. Others have taken the opposing view, however, and attribute increases in psychological test scores to headache pain parameters (Andrasik et al., 1982a; Dalessio, 1980; Philips, 1976; Sternbach et al., 1980). Support for this view is provided by studies finding that headache patients do not differ from chronic pain patients on the MMPI (Andrasik et al., 1982a; Watson, 1982) and studies that have found the frequency of headache activity, regardless of diagnosis, to be related to psychological disturbance (Drummond, 1983; Harper & Steger, 1978; McAnulty, 1984). Studies directly addressing the cause-effect issue with personality characteristics and headaches are lacking. However, there has been research addressing the relation between emotional states and headaches, which is reviewed in a

later section of this manuscript.

Psychobiological formulations. In recent years, behavioral scientists have attempted to integrate the psychological and biological findings pertaining to headache into a psychobiological conceptualization. Such formulations have been offered to explain both migraine and muscle-contraction headaches. Cinciripini, Williamson, and Epstein (1980) have outlined possible relations among physiological, behavioral-environmental, and behavioral-cognitive variables, in their "Biobehavioral Theory", that could lead to the development and maintenance of migraine headaches. Physiological variables include those indicative of autonomic arousal (e.g. heart rate, respiration rate, electromyographic activity, and vasomotor activity).
Behavioral-environmental factors include the variety of potential precipitants, described earlier, such as certain foods, alcohol, light, and noise, as well as consequential conditions that may reinforce, and thus increase, an individual's likelihood of reporting head pain. Potential reinforcers could include increased attention from others and/or a reduction in the individual's usual familial and occupational responsibilities (i.e., secondary gain). In this way, pain reports may be increased or maintained with little or no actual physiological involvement (Cinciripini et al., 1980; Demjen & Bakal, 1981). Behavioral-cognitive factors include the individual's thoughts, interpretations

of environmental events, and coping strategies used in response to stress. Cinciripini and associates propose that stressful circumstances can produce strong cognitive and emotional reactions and may increase the probability for a headache in those physiologically predisposed.

Bakal (1975) has also proposed a "Biopsychological" model for migraines. His model postulates that there is a predisposition for the physiological changes observed in migraine headache. According to Bakal, this predisposition may have been produced from an inherent variability or reactivity in the physiological responses (implicated in migraines) in response to psychological stress. Further, he suggests that with repeated exposure to the same stressful events, these same events may no longer be perceived as stressful but, through classical conditioning, may still potentiate the abberrant physiological responses. The main assumption of both of the above thoeries is that when the individual is under stress, the physiological processes implicated in migraine are activated. Similar psychobiological formulations have been offered, by the above authors, to explain muscle-contraction headaches (Bakal, Demjen & Kaganov, 1981; Epstein & Cinciripini, 1981). Again, precipitating stressful events, physiological and cognitive and emotional reactions, and behavioral and environmental consequences are the key components. The main difference between migraine and muscle-contraction headaches,

according to these formulations, lies in the different physiological response systems believed to be activated.

The main difference between the psychodynamic and the psychobiological formulations for headaches are subtle. Psychodynamic theorists believe that constitutional personality differences determine which specific physiological system is activated, when an individual is exposed to stress, to produce the different headache types. Psychobiological theorists believe that constitutional physiological differences exist, which are activated when the individual is stressed, to produce the different headache syndromes. The two approaches seem to agree, however, on two basic points: (a) that stressful situations are important precipitating factors in headaches; and (b) that stress can produce abberrant psychological and physiological reactions in headache sufferers. The evidence available on these two points are critically reviewed in the next section.

Stress and Headaches

The biological, psychological, and psychobiological theories of headache have all implied that stress plays an important role in headaches. Numerous researchers have also provided information, mostly from relatively large survey studies, suggesting that a stress-headache relation exists. Stress is frequently reported as a major contributing factor in both tension headaches (Ad Hoc Committee, 1962; Drummond, 1983; Friedman, 1964, 1979;

Friedman et al., 1954; Kudrow, 1979) and migraine headaches (Dalsgaard-Nielsen, 1965; Drummond, 1983; Friedman et al., 1954; Henryk-Gutt & Rees, 1973; Parnell & Cooperstock, 1979; Selby & Lance, 1960). For example, one early survey concluded emotional factors to be contributory in all of their 2,000 cases of chronic muscle-contraction and migraine headache (Friedman et al., 1954). Various studies have estimated from 54 to 68% of migraine headaches are precipitated by emotional stress (Dalsgaard-Nielsen, 1965; Henryk-Gutt & Rees, 1973; Selby & Lance, 1960). In addition, Henryk-Gutt and Rees (1973) found that one-half of their migraine headache sufferers reported the initial onset of their headache problem to coincide with a period of emotional stress. Recently, Feuerstein, Bush, and Corbisiero (1982) found that 63.6%, 87.5%, and 100% of their migraine, muscle-contraction, and mixed headache subjects, respectively, reported stress as a precipitating factor. Further, Featherstone and Beitman (1983) concluded that 72% of their migraine sufferers reported that emotional stress was present in their personal lives.

Although the studies reviewed above strongly suggest that stress is related to both migraine and tension headaches, several qualifying points deserve mention. First, there is more evidence available to support this perspective for migraine sufferers than for tension headache sufferers. As others have noted (e.g. Beaty &

Haynes, 1979; Ziegler, 1978), this is surprising given that the Ad Hoc Committee (1962) definition states that tension headaches occur "usually as part of the individual's reaction during life stress" (p. 128). Second, all of the above studies obtained their results from retrospective, nonstandardized and often, unstructured interviews. In these studies, participants were asked to report the factors they felt have occurred and/or have precipitated their headaches in the past. The percentage of subjects reporting that stressful events were/are present or that emotional stress has, on some occasions, precipitated their headaches is reported. Consequently, the findings of these studies may reflect only the percentage of subjects who perceive that there is a stress-headache relation for themselves or may reflect a percentage based on investigators' judgments of the presence of stress. For example, only 72% of the subjects assessed by Friedman et al. (1954) felt stress contributed to their headaches, far less than the 100% figure determined by these authors. Similarly, Featherstone et al. (1983) found that only 44% of their migraineurs related their headaches to emotional stress although 72% reported that emotional stress was currently present. Selby and Lance (1960) found that 33% of their 388 headache sufferers did not consider stress to be a precipitant. A third qualifying point is that none of the above studies reported information on the amount of stress

present in a comparison population. As a result, we cannot evaluate whether or not headache sufferers experience more stress in their lives than other groups. Finally, these studies did not attempt to determine the strength or nature of the relation between stress and various patterns of headache activity (i.e., frequency, intensity, duration). Consequently, the results of past research do not evaluate whether or not a greater amount of life-stress results in more headaches, and/or headaches of greater intensity and/or longer duration.

Current Methods for Measuring Stress

Over the past 15 to 20 years several theoretical and methodological advances have been made within the field of stress. As a result, researchers have begun to conceptualize and measure stress more pragmatically. Most theorists conceptualize stress as either some event or stimulus that impinges on and causes some reactive change in the individual, "stimulus" theories, or as the reaction or response of the individual as a consequence of the occurrence of some stressful event or stimulus, "response" theories (Derogatis, 1982). According to response theories, it is the emotional and physiological response of the individual, to events in the environment, that define the presence of stress. Researchers working within this framework tend to use measures of disrupted functioning including symptom inventories (e.g., SCL-90R), scales measuring negative

emotional states (e.g., Beck Depression Inventory), and physiological assessments for measuring stress. Stimulus-oriented theorists, on the other hand, define stress in terms of the objective nature of the stimulus that impinges on the individual. According to this framework, the task in measuring stress is to obtain an accurate assessment of those aspects of the environment that are disorganizing or demanding for the individual. Life-events scales (e.g., Social Readjustment Rating Scale) are typically used in this line of research. More recently an "interactive" model of stress has been evolving (e.g., Lazarus, 1966; Lazarus & Folkman, 1982). According to this view, mediational processes, involving individuals' evaluations and judgments, determine the extent of their reactions to environmental events. These evaluations and judgments are thought to be influenced by an individual's genetic make-up, past experiences, personality traits, attitudes, internal physiological states and the like. Thus, an individual's perception and evaluation of an environmental event reflect these mediating factors and subsequently influence his or her response. From this point of view, the task in measuring stress not only involves assessing the objective nature of the environmental stimuli and an individual's responses, but also involves assessing the individual's subjective evaluation or judgment of the stimuli. Headache researchers have attempted to measure stress from all of

these competing yet complementary vantage points. Methods that have been used for measuring the stress evident in headache sufferers are: (a) assessing their physiological responses to stressors presented in the laboratory; (b) assessing their subjective reports of distress and their emotional states in response to laboratory stressors and stressful life-events; and (c) quantifying the amount/number of stressful life-events to which they have been exposed.

Physiological responses to laboratory stressors. One method frequently used to assess the stress experienced by headache sufferers has been to assess their physiological reactions (e.g. heart rate, skin resistance, electromyographic activity, and vasomotor functioning) to standard stressors presented in a controlled laboratory setting. Typical stressors presented to subjects include orienting and startling tones, mental arithmetic tasks, mock "intelligence" tests, mental imagery, and the inflation of an occlusion cuff.

There is no question that stressful stimuli, presented in controlled laboratory settings, produce changes in physiological responses indicative of autonomic arousal in both headache and nonheadache control populations (Anderson & Franks, 1981; Andrasik et al., 1982b; Bakal & Kaganov, 1977; Brantley, 1980; Cohen, Rickles, & McArthur, 1978; Feuerstein et al., 1982; Gannon, Haynes, Safranek, & Hamilton, 1981; Haber et al.,

1985; Passchier, van der Helm-Hylkema, & Orlebeke, 1984; Philips, 1977; Pozniak-Patewicz, 1976; Sturgis, 1981; Vaughn, Pall, & Haynes, 1977). Further, most often, no differences in the magnitude of physiological reactivity to laboratory stressors are found between headache and control groups (Anderson & Franks, 1981; Andrasik et al., 1982b; Bakal & Kaganov, 1977; Brantley, 1980; Gannon et al., 1981; Haber et al., 1985; Passchier et al., 1984; Sturgis, 1981). Only one study found classic migraineurs to evidence greater autonomic reactivity than controls, and only during an atypically long and unique cognitive stressor (Cohen et al., 1978). Most often, when differences are found between groups, they reflect different patterns of physiological responding, providing evidence for response stereotypy, the notion that physiological responses will be the greatest in those systems believed to be related to the patients' somatic complaints, (see Lacey & Lacey, 1958; and Malmo & Shagass, 1949), rather than differences in the reactivity of responses (Bakal & Kaganov, 1977; Cohen et al., 1978; Cohen, Williamson, Monguillot, Hutchinson, Gottlieb, & Waters, 1983; Gannon et al., 1981). No differences in patterns of physiological responses have been found equally often, however (Anderson & Franks, 1981; Andrasik et al., 1984; Brantley, 1980; Sturgis, 1981).

In general, the above findings indicate that: (a) laboratory stressors do activate physiological responses

in headache groups; (b) headache groups do not consistently differ from controls in the magnitude of these responses (i.e. they do not physiologically overreact to stress); and (c) when differences between groups are found, they usually refer to different patterns of physiological responding (i.e., greater magnitudes in some responses and lesser magnitudes in others).

Subjective emotional responses to laboratory stressors. Research focusing on the psychological functioning of headache sufferers, particularly psychodynamic theories, suggest that headache sufferers may overreact to stressful events. The laboratory studies reviewed above fail to support the notion that headache sufferers physiologically overreact, as compared to controls, to stressors presented in the laboratory. Considering the large number of studies examining physiological reactivity to laboratory stressors, surprisingly, only three of these studies have examined their subjects' ratings of subjectively experienced stress in response to these stressors (Brantley, 1980; Feuerstein et al., 1982; Passchier et al., 1984) and none examined their subjects' emotional responses. In contrast to expectations, none of these three studies found headache subjects to report experiencing more subjective distress, in response to laboratory stressors, than control subjects. Passchier et al. (1984), however, did find headache subjects, both migraine and muscle-contraction

sufferers, to report more subjective tension than controls during relaxing imagery and during recovery-from-stress periods.

Additional evidence related to the issue of whether or not headache sufferers overreact to laboratory stressors can be gleaned from studies examining pain thresholds in headache and control subjects. Gannon et al. (1981) found that 11 of 16 migraine and 10 of 13 tension headache sufferers, as compared to only 6 of 15 control subjects requested the premature termination of a physical stressor, inflation of an occlusion cuff. This suggests that headache subjects may subjectively experience the same physical stressor more intensely than controls. Further support for this notion has recently been provided by Klein (1983). In this study, ten migraine and ten "nonmigraine" headache sufferers were compared on their self-reported preferences for situations of various levels of stimulation. The findings indicated that the migraine subjects preferred situations and activities with less intense levels of stimulation than those preferred by the comparison group. Based on this finding, Klein (1983) concluded that migraineurs are stimulus intensity "augmenters" and may be hypersensitive to environmental stress. Contradictory evidence is provided by others, however, who have found no differences between headache and control subjects in pain tolerance (Feuerstein et al., 1982; Haynes, Gannon, Cuevas, Heiser,

Hamilton & Kafranides, 1983; Martin & Mathews, 1978). In fact, Martin and Mathews (1978) found a slightly, although not statistically significantly, higher threshold for their headache subjects. Further, Price and Blackwell (1981) found migraineurs to report less subjective stress, than controls, in response to a stressful film.

Brantley (1980) compared eight migraine and eight tension headache sufferers to eight control subjects on both subjective and physiological responses to psychological stressors presented in the laboratory. Subjects were given two physiological test sessions, one during a subjectively "high" stress day and the other on a subjectively "low" stress day. He found that while tension headache and control subjects reported markedly higher subjective stress in response to stressful laboratory stimuli than to relaxing laboratory stimuli, the migraine subjects did not make this discrimination and reported consistently high ratings of subjective stress across all the stimuli. In addition, the migraineurs' subjective ratings were significantly higher than the ratings of the other two groups. Further, the physiological responses of all subjects reflected autonomic arousal in response to stressful stimuli relative to relaxing stimuli. An interesting difference, however, between the migraine and muscle-contraction subjects was observed. Although the muscle-contraction subjects maintained high autonomic arousal to all

stressful stimuli, the migraine subjects evidenced their greatest physiological arousal during "feel" stress stimuli as compared to "think" stress stimuli. Brantley concluded that these results suggest that tension headache sufferers react both emotionally and physiologically to the mere presence of stressful circumstances. Migraine headache sufferers, however, produce their most marked physiological arousal in response to stimuli possessing an emotional component. The results obtained by Brantley (1980) suggest that future studies investigating the relation between stress and headaches need to assess not only the subjective stress experienced in response to stressful circumstances, but also the emotional responses of individuals as well. Taken together, these studies suggest that various headache and control groups may indeed experience stressful events differently, however, no consistent trend in the direction of this difference has yet emerged. This area of research deserves further attention.

Problems with measuring stress in the laboratory. There are problems inherent with the use of laboratory stressors that may contribute to the inconsistent results observed above. Several researchers have noted that laboratory stressors may be inadequate for determining an individual's response to stress (e.g. Adams et al., 1980; Blanchard & Andrasik, 1982; Haber et al., 1985). Specifically, they point out that the stressors used in

these studies are generally mild and of short duration (for obvious ethical reasons) and may not be perceived as personally stressful across subjects. Further, the novel nature of the typical experimental stressor and the artificiality of the environment in which they are presented limits the generalization of findings to the "real" world. Whether research investigating the stress process is most appropriately conducted in the laboratory or in the field is a current topic in debate (Laux & Vossel, 1982). Laux and Vossel suggest that laboratory studies are more appropriate for generating predictions to be subsequently checked, against real-world events, through field studies. Controlled laboratory studies appear to be more appropriate for examining if and how stressful events trigger the pathophysiological mechanisms implicated in headaches while field studies appear to be more appropriate for examining the nature of the relation among real-world stressful events, affective states and headache activity. For example, these laboratory studies do not tell us when a headache sufferer is most likely to experience a headache. Is it on the day s/he experiences a high level of subjective stress or an emotionally arousing situation (or both) or is it on the one or more days that follow? A field study, where individuals monitor their subjective stress levels, affective states, and headache activity across several days is a more appropriate approach for addressing this issue.

Although current conceptualizations of the stress process emphasize the importance of an individual's interpretation of a stressful event in determining the nature of the emotional and physiological responses produced, there is an apparent dearth of evidence available with headache populations. In spite of this, the existence of a relation between emotional states and migraine headaches has been widely accepted, mostly as a result of clinical observations (Dalessio, 1980; Sacks, 1981). Two recent investigations have provided some empirical support for the notion that affective states may be important in headaches. Harvey and Hay (1984) observed affective states and headache activity in 10 migraine sufferers over a 30-day period. Their subjects reported a positive mood change on the day preceding a headache attack and a worsening of mood on headache days on the Depression Adjective Checklist (DACL) and a visual analogue scale measuring mood. In a quasi-field study, Feuerstein, Bortolussi, Houle, & Labbe (1983) measured physiological responses and state anxiety, daily, over five days (4 days preceding a headache and the day of the headache) with 12 migraineurs. They found that the anxiety score four days preceding headaches significantly correlated (r = .62) with increased autonomic variability 3 days prior to the onset of headache. In addition, the strongest relation they observed was between anxiety scores on headache days and arterial blood volume

variability one day prior to headaches. They suggest that their results indicate that anxiety may be associated with the onset of autonomic dysregulation, which may be apparent up to 96 hours prior to the occurrance of a migraine headache.

Neither of these studies examined the possible role that stressful life-events played in producing these mood changes. They underscore, however, the importance of measuring daily affective states in relation to headache activity. Unfortunately, very few field studies, investigating the effects of stressful life-events on headache, have been conducted. The few studies that have looked at stressful events and affective states outside of the laboratory, with headache populations, have provided modest but positive results.

Headache Activity and the Stressful Life-Events Approach to Measuring Stress

The majority of research approaching stress from the "stimulus" model has used the Schedule of Recent Experience (SRE) (Hawkins, Davies, & Holmes, 1957) and the subsequent Social Readjustment Rating Scale (SRRS)(Holmes & Rahe, 1967) to measure stress. These scales ask respondents to indicate which, if any, major life-events they experienced during a previous, predetermined period of time, usually one year. Typical of the events included on these scales are: loss of job, divorce, pregnancy, and death of spouse. The scales provide two types of scores,

one reflecting the number of events that occurred and one reflecting the amount of change or adaptation required of the individual to adjust to the events. Whereas the previously used interview methods were nonstandardized, subjective, and mainly used to determine if stress was present, the life-events scales are standardized, objective, and can be used to quantify the identified stressors. Research with these scales generally find that a modest but significant relation exists between stressful life-events and the onset or exacerbation of numerous medical conditions and psychological disorders (see Dohrenwend & Dohrenwend, 1978; Rabkin & Streuning, 1976; for reviews). Only within the last five years, however, have these life-events scales been used with headache populations.

To date, only three articles assessing stress and its relation to headaches with these measures have appeared in the literature. All three studies used the SRRS or a modified version of it (Andrasik et al., 1982b; Andrasik & Holroyd, 1980; Blanchard et al., 1984). Two of these assessed the amount of life-stress present and one examined the relation between life-stress and headache activity. Andrasik and Holroyd (1980) compared 39 tension headache sufferers to 23 control subjects with a version of the SRRS modified for students. Their results indicate that tension headache sufferers report no more stressful life-events than controls. In a larger study, Andrasik et

al. (1982b) compared 99 subjects suffering from either migraine, tension, mixed, or cluster headaches to 30 matched nonheadache controls on several psychological measures, including the SRRS. They used separate scores from the SRRS for each of three time periods prior to their assessment session; 0-6 months, 7-12 months, and a combined score for 0-12 months. As in the previous study, no differences were found among any of the groups on any of the stress scores. In a more recent article, Blanchard et al. (1984) used their same data set to examine the relations between their various measures from the psychological, biological, and socio-demographic realms and headache activity. They performed separate stepwise multiple regressions with the scores from each realm, both separately and combined, to predict headache activity within each group. When the scores from all three realms combined were entered into these computations, none of the scores from the SRRS accounted for a significant amount of the variance in headache activity for any group. When only the psychological scores were entered, however, the 7-12 month score significantly entered the equation for the migraine group. For this group, this score accounted for more of the variance in headache activity (16%) than any other score that met statistical criteria to be entered into the equation, including scores from scales 6 and 7 of the MMPI and trait anxiety. In summary, two studies have not found headache sufferers to report

experiencing more stressful life-events than nonheadache controls. One study, however, found the amount of stressful life-events to be significantly related to migraine but not tension or mixed headache activity. Andrasik and Holroyd (1980) concluded that because their headache subjects were exposed to no more stress than their control subjects, that headache sufferers may "overreact" to life stress. Although this conclusion is consistent with those of others (e.g. Dalsgaard-Nielsen, 1965; Henryk-Gutt & Rees, 1973), there are several problems apparent with studies using the SRRS as a life-event measure, specifically, and with studies using life-events measures, in general, that need to be remedied before firm conclusions can be drawn.

Problems with life-events scales as measures of stress. As previously mentioned, "interaction" theorists argue against a simple stimulus-oriented model of stress to explain the complex stress process (Lazarus & Folkman, 1982). Within the stress literature, long-standing methodological issues, concerning the measurement of stress with life-events scales, have been debated. These issues involve: (a) whether only the number of events endorsed as having occurred should determine the score or whether the events should be weighted to reflect their relative impact upon the individual; (b) whether or not the weighting of the events should be normative or idiographic; and (c) whether or not

positive events should be assessed in addition to more aversive events (Cleary, 1980; Rabkin & Streuning, 1976). Although there is still some disagreement, the evidence suggests that individuals rate the same events differently, that some events are considered more stressful than others, and that it is the occurrence of the more aversive events that correlates with negative affect and symptoms (Sarason, Johnson & Seigel, 1978; Vinokur & Selzer, 1975; Zeiss, 1980). As previously mentioned, headache researchers have used the SRRS to assess life-events. In its original form, the SRRS is gradually becoming outdated because its scores are based on a normative weighting system and, also, do not reflect the relative aversiveness of the events, that is, it treats all events as if they have an aversive effect. For these reasons Sarason, Johnson and Seigel (1978) developed their Life Experiences Survey (LES) which allows idiographic, subjective ratings of the relative aversiveness of life events. Although headache sufferers have been postulated to overreact to life-stress, the scoring system of the SRRS assigns the same weight to the same event across individuals which does not allow a direct test of this hypothesis.

Although theoretical and methodological advances have been made in the field of life-stress assessment, since the efforts of Holmes and Rahe (1967), most of these advances have not been incorporated into research with

headache populations. Certain aspects of migraine and muscle-contraction headaches also make these life-events measures of only limited usefulness for studying stress-headache relations. First, most individuals' headache problems will vary somewhat, in frequency, intensity, and duration, from week-to-week. Second, headache theorists presume that stressful events, when they are triggering factors, probably occur anywhere from a few hours, for tension headaches, up to a few days, for migraine headaches, before a particular headache bout. Subsequently, researchers are faced not only with the task of assessing life-stress but also with the task of explaining exacerbations and remissions in headache activity.

In addition to the problems with the SRRS as a measure of stress, there are problems and subsequent limitations with the use of these life-event measures, in general (see Cleary, 1980; Kanner, Coyne, Schaefer & Lazarus, 1981; Dohrenwend & Dohrenwend, 1978; Pearlin, 1982; Perkins, 1982; and Rabkin & Streuning, 1976, for recent reviews). First, these scales involve rather long-term retrospective reports on the respondents' part. This problem is not as much one of the respondents' ability to remember whether or not an event occurred as it is one of the respondents' ability to provide accurate accounts of the perceived stressfulness of the event at the time when it occurred. Second, these scales do not

give much weight to the amount of intervening time between the occurrance of an event and the onset or exacerbation of symptoms (Cleary, 1980). Failing to specify this intervening time can be costly since, as Dohrenwend and Dohrenwend (1978) have concluded, the lack of this information can impede the ability, of investigators, to move from correlational to causal statements. The establishment of temporal contiguity between the occurrence of stressful events and the onset or exacerbation of a physical condition is a major criterion for using the diagnostic category "Psychological Factors Affecting Physical Condition" in the Diagnostic and Statistical Manual of Mental Disorders (DSM-III, American Psychiatric Association, 1980). Headache is offered, by the DSM-III, as an example of one condition that could fit into this diagnostic category. The DSM-III suggests that "repeatedly" demonstrating that a physical condition occurs or is exacerbated following stressful events can strengthen the case that a stress-disorder relation exists. The most widely used life-events scales (SRE, SRRS, LES) are designed to be administered at most, once every 6 months. As a result, the scores they provide cannot examine whether the occurrence of these events is related to any one particular headache bout or to observed exacerbations and remissions of symptoms. Brown (1981) has suggested that the strength of the causal stress-disorder relation could be more accurately

estimated if events and pathology were assessed as often as on a weekly basis. In summary, the usefulness of these life-events scales is limited to: (a) the objective assessment of the number of life-events to which individuals have been exposed during previous months; (b) the quantification of the individuals' perception of the stressfulness of these events (if the scale allows idiographic subjective impact ratings); and, only to a limited extent, (c) providing an estimate of the influence of life-events on present headache activity. Some very recent advances in life-events measurement have the potential of rectifying several of the limitations of these measures.

Recent researchers (Brantley, Waggoner, Jones, & Rappaport, in press; DeLongis, Coyne, Folkman & Lazarus, 1982; Kanner et al., 1981) have suggested that measures such as the SRRS measure "major" life events and do not adequately assess the possible universe of stressful events. Kanner et al. (1981) suggest that the assessment of relatively minor, day-to-day "hassles", for example, arguments, social pressures, and job strains, may increase the strength of stress-disorder correlations. They have found the occurrence of such minor events to significantly correlate with negative affective states and to account for a significant portion of the variance in the presentation of psychological and physical symptoms over and above the variance accounted for by major life-events.

Although the Hassles Scale appears to be an adequate measure of minor stressful events, it only provides a monthly measure of these events. The Daily Stress Inventory (Brantley et al., in press) has taken the assessment of minor stressful events a step further. This inventory assesses minor stressful events on a daily basis and has been found to be significantly correlated with scores from the Hassles Scale. Conceptually, these minor events could occur in closer temporal proximity to any given headache bout than a previous major life-event. Consequently, major life-events scales may be more appropriate for predicting the initial onset of or the more long-term tonic (i.e., year-to-year) fluctuations in headache activity whereas a daily measure may be more appropriate for predicting the short-term periodic exacerbation or remission of headaches.

Based on the above review of the problems involved in the assessment of stressful life-events, researchers need to: (a) use a major life-events scale that allows the separation of aversive and positive/beneficial events, that allows individualized ratings of the relative aversiveness of the events, and that allows some specification of when, during the previous year, the events occurred; (b) assess relatively minor day-to-day events in addition to major life events; and (c) use a measure of minor stressful events that allows the separation of aversive and positive events, that allows

individualized ratings of the impacts of the events, and that can be completed as often as on a daily basis.

There are a few studies available that take into account some, but not all, of these recommendations. These studies are discussed below.

More Comprehensive Studies Examining the Role of Stressful Life-Events in Headache

The first study to examine both the amount of stress present in the lives of headache sufferers and the relation of ongoing life-stressors to headache activity was conducted by Henryk-Gutt & Rees (1973). As was previously mentioned, these investigators assessed the presence of major stressful life-events with an unstructured interview. To their credit, they also had their 18 migraineurs monitor their headache occurrences for two months along with "any unusual or special events coinciding in time with the attacks." As a result of this procedure, they concluded that 54% of all of their migraineurs' attacks coincided with emotional stress. From these findings, they suggest that migraine sufferers are predisposed by constitutional factors to experience a greater than average reaction to a given quantity of stress. Although they assessed major and minor stressful events, their conclusions are limited by a number of problems. In addition to the problems associated with the subjective methods they used for establishing the presence of stress, several other problems are apparent: (a) they

did not have subjects provide subjective ratings of their reactions to the stressful events; (b) the stress was not quantified; (c) the types of stressful events (i.e. major vs. minor vs. both) were not specified; (d) they did not specify how close in time the stressful events actually "coincided" with the headache attacks; and (e) they did not include a comparison population. As a result this study was not designed to determine the quantity of stress present, subjects' reactions to the stress, or the strength of the stress-headache relation.

More recent studies have also incorporated some of the advances made in the stress research methodology for the assessment of stressful life-events with headache populations (Andrasik et al., 1984; Brantley, 1980; Pratt et al., 1982). Andrasik and his associates (1984) modified the SRRS to allow the computation of stress scores that reflected two separate time periods, 0-6 months and 7-12 months, prior to their assessment session. In addition, they related these scores to current headache activity by using multiple regression analyses. Their results revealed, as a result of these two methodological advances, a significant relation between the 7-12 month stress score and their index of headache activity. In another study, Pratt et al. (1982) modified the SRE to provide three scores: (a) a total score to reflect the amount of life stress present during the previous 12 months; (b) a recency score to reflect how long ago each

event occurred; and (c) subjective ratings of the stressfulness of the events. Although the headache groups did not differ from each other or from the control subjects on these scores taken separately, a multiple discriminant analysis performed with these scores revealed that the recency score significantly discriminated between the groups. Specifically, they found migraineurs to report their stressors to be more remote in time than the tension headache sufferers. Interestingly, their control subjects scored slightly higher than the headache groups on the total stress scores and on the subjective ratings of stress. These results contrast with earlier suggestions that headache sufferers overreact to a given quantity of life stress.

In another, more comprehensive study investigating life-stress and headaches, Brantley (1980) not only assessed major life-events with a more advanced instrument, but also, assessed relatively minor stressful events with an objective instument that also allowed subjective ratings of the stressfulness of these events. He compared eight migraine and eight tension headache sufferers to eight control subjects on responses to the Life Experiences Survey (LES), and on responses to an unstandardized objective measure of minor daily stressful events, the Daily Stress Record. In this study, the participants monitored their headache activity along with stressful events and a Likert-type rating (0-10) of their

overall subjective stress daily for three weeks. As in the two previously discussed studies, the headache groups did not differ from the control subjects in the number of or subjective reactions to major stressful life-events nor on scores from the daily measure of minor stressful events. However, the migraine group tended to report the most subjective daily stress, followed by the tension group, which was followed by the control group. It seems likely that a significant difference would emerge, from this tendency, with larger sample sizes. In addition, the daily stress scores did not significantly correlate with the headache activity of any group. Of interest, however, is the fact that the "nonheadache" subjects in this study did report having an occasional headache. When the correlations between daily stress scores and the headache activity of each group were transformed to z scores and compared, the headache groups', particularly the tension headache group, headaches were significantly more related to daily stress than were the control group's headaches. This finding supports the notion that life-stress is more likely to be related to headaches in those predisposed to have headaches than to headaches in those not so predisposed. Brantley's conclusions are compromised only by his small sample sizes and the use of a daily stress measure that, although it was objective, had not yet been standardized or validated. In spite of these limitations, these findings suggest that future

studies should not only assess major life-events but also examine the influences of daily stressful events and levels of perceived stress in headache sufferers.

The Present Study

Stress is frequently reported as a major contributing factor in both tension headaches (Ad Hoc Committee, 1962; Drummond, 1983; Friedman, 1979; Kudrow, 1979) and migraine headaches (Dalsgaard-Nielsen, 1965; Drummond, 1983; Friedman et al., 1954; Henryk-Gutt & Rees, 1973; Selby & Lance, 1960). The general conclusions of these studies are that stress can precipitate headache attacks and that headache subjects tend to "overreact" (emotionally and physiologically) to stress. Much confusion over the definition of "stress" is evident in the early studies. Early attempts at gathering evidence for a stress-headache relation were positive but relied on retrospective, nonstandardized, and unstructured interviews, as well as, subjective evaluations. As the methodology for measuring stress becomes more refined and new theories explaining the stress process (i.e. stimulus, response, and interaction theories) have evolved, the hypothesized stress-headache relations are becoming more complex and the evidence more equivocal.

One current method used by headache researchers for measuring stress involves evaluating psychophysiological and subjective responses to stressors presented in the laboratory. These studies attempted to demonstrate that

headache sufferers, as compared to controls, overreact to stress. Although these studies demonstrate that headache subjects physiologically react to laboratory stressors, the responses of headache subjects have not been found to consistently differ from those of control subjects (e.g., Anderson & Franks, 1981; Andrasik et al., 1982b; Gannon et al., 1981). Few studies have investigated differences in subjective emotional responses to laboratory stressors. In spite of this, the available evidence suggests that various headache and control groups may subjectively experience stress differently but the results are often contradictory (Brantley, 1980; Passchier et al., 1984; Price & Blackwell, 1981). Problems with the laboratory method have been cited as being responsible for negative results.

The laboratory method has been criticized because of: (a) the inadequate nature of the stressors employed; (b) the artificiality of the laboratory environment; and (c) the inability of these designs to examine the nature of the stress-headache relation as it occurs in the real-world (Adams et al., 1980; Laux & Vossel, 1982). Several field studies have provided evidence that headache activity may very well be related to subjective emotional reactions and stressful life-events (Brantley, 1980; Feuerstein et al., 1983; Harvey & Hay, 1984). Migraine headaches may occur up to 4 days following an increase in anxiety (Feuerstein et al., 1983). The relation for

muscle-contraction headache sufferers has been relatively unexplored but these headaches are generally believed to occur immediately, for example, within a few hours, following emotional stress (Ad Hoc Committee, 1962). The relations between stress and different headache types, although hypothesized to be different, have not been compared.

Another method used for measuring the stress associated with headaches involves the use of life-events scales such as the Social Readjustment Rating Scale (Holmes & Rahe, 1967). These scales offer an objective method for quantifying real-world stressful life events and have been found to correlate with numerous medical conditions and psychological disorders (Dohrenwend & Dohrenwend, 1978; Rabkin & Struening, 1976). Only three studies using these scales have appeared in the headache literature (Andrasik et al., 1982a; Andrasik et al., 1982b; Andrasik & Holroyd, 1980), and none found headache groups to score differently than control groups on these scales. These studies lead one to believe that, if headache subjects and controls have experienced the same amount of stress then headache subjects must overreact to stress because they have headaches, circular reasoning. Unfortunately, the life event scales used in these studies do not evaluate an individual's subjective reaction to the life-events because their scoring systems are purely normative and not idiographic. In addition, because these

scales involve rather long-term retrospective reports, they cannot examine the nature of the relation between the occurrence of stressful events and when a particular headache will occur. Clearly, these scales are of only limited usefulness for studying short-term stress-headache relations. Recent stress theorists suggest that major life-event scales need to: (a) allow for subjective idiographic ratings of the stressfulness of the events; (b) obtain an estimation of when the events occurred; and (c) assess relatively minor stressful events in addition to major life-events (DeLongis et al., 1982; Kanner et al., 1981; Sarason, Johnson, & Seigel, 1976). Several recent studies have incorporated some, but not all, of these suggestions in studying the stress-headache relation and obtained some promising results (i.e., Andrasik et al., 1984; Brantley, 1980; Pratt et al., 1982).

The above review suggests that a more comprehensive study of the stress-headache relation is needed. In an effort to combine and improve upon methods previously used and, therefore, to increase our understanding of the nature of the role of stress in headaches such a study needs to: (a) use a field method to increase the generalizations of findings to real-world events; (b) assess stress with a major life-events scale that allows individual subjective ratings of the events and allows some specification of when the events occurred; (c) assess relatively minor, day-to-day stressful events with a

measure that allows individual subjective ratings of the events and that can be administered as often as on a daily basis; and (d) assess daily emotional states. No study to date has concurrently assessed daily headache activity, daily stress levels, and daily emotional states in conjunction with major life-events.

The present study examined the relation between stressful life-events and headache activity in several headache populations. This study not only examined the relation(s) between stress and headache activity by traditionally used methods, that is, by using a major life-event scale, but will also departed from the previously used methods, thereby overcoming many of the methodological problems outlined above, by concurrently assessing daily headache activity, daily relatively minor stressful events, daily perceived stress, and daily emotional states. Specific questions this study addressed included:

1. a. Do the groups differ in the number of negatively rated major life-events that members report having experienced during the past year?

   b. Is the amount of present headache activity related to the number of negatively rated major life-events?

   Hypotheses: (a) Headache and control groups are not expected to differ in the number of negatively rated major life-events they report; and (b) the

number of these events is not expected to be related to headache activity.

2. a. Do the groups differ in the number of present, relatively minor, daily stressful events they experience over the 28 days of the study?

   b. Is the amount of present headache activity related to the number of daily stressful events?

   Hypotheses: (a) Headache and control groups are not expected to differ in the number of daily stressful events they report, and (b) the number of these events is not expected to be related to headache activity.

3. a. Do the groups differ in the degree of their subjective reactions to major and/or minor stressful life-events?

   b. Is headache activity related to the degree of these subjective reactions?

   Hypotheses: (a) The headache groups are expected to experience more intense reactions to stressful events than are control subjects; and (b) the intensity of their subjective reactions is expected to be related to headache activity.

4. Does combining the assessment of major life-events with the assessment of minor life-events increase the prediction of present headache activity?

   Hypothesis: Combining the assessment of

major and minor stressful events is expected to significantly increase the prediction of headache activity over that from the assessment of major life-events alone.

5. a. Do the groups differ in the intensity and/or type of their present daily affective states?
   b. Is the intensity and/or type of daily affective state related to the number or amount of major and/or minor stressful life-events?
   c. Is the intensity and/or type of daily affective state related to present headache activity?

Hypotheses: (a) Headache groups are expected to experience greater anxiety, depression, and hostility than controls; (b) although significant between-headaches groups differences are not expected, past research findings suggest that the headache groups should fall on a continuum with the tension headache group experiencing the most intense affective response followed by the mixed and then the migraine group; (c) the intensity of the affective responses is expected to be positively related to the amount/number of daily stress(ors) with the headache subjects evidencing significantly more intense responses than the controls; and (d) the type of affective response is not expected to

be related to headache activity nor to any particular headache diagnosis.

6. Do the headache groups differ in the amount/number of daily stress(ors) they experience one and two days preceding their headache as compared to the amount/number of daily stress(ors) they experience on headache days?

   Hypothesis: Significant within-group differences across days are expected with respect to both the number of stressors individuals' experience and their subjective reactions to stress. Specifically, tension headache sufferers are expected to experience their greatest amount/number of stress(ors) on headache days while migraine sufferers are expected to experience their greatest amount/number of stress(ors) one or two days prior to their headaches.

7. Do the headache groups differ in the intensity or type of affective state they experience one and two days preceding their headache as compared to headache days?

   Hypothesis: Although significant

between-groups differences in the intensity of and type of affective response are not expected, within-group differences in the intensity of affective responses are expected across headache and nonheadache days. Specifically, tension headache sufferers are expected to experience their most intense affective states on headache days while migraine sufferers are expected to experience their most intense affective states one or two days prior to their headaches.

Method

Subjects

Twenty migraine, 20 muscle-contraction, 20 mixed migraine and muscle-contraction, and 20 control subjects participated in the present study. The age and sex characteristics of the groups are presented in Table 1. These subjects participated in this project as part of a larger, ongoing project investigating various causes of headaches. The participants in this larger project were recruited by means of a local newspaper article, circulated in the Baton Rouge, LA and surrounding area, that solicited individuals to become involved in a study investigating the causes of headaches, and by referrals from private physicians in the community. Upon an individual's initial phone contact s/he provided the information requested on the Telephone Screening Form

Table 1

Age and Sex Composition of Diagnostic Groups

Group	Male	Female	M Age
Control	7	13	35.75
Migraine	2	18	41.10
Mixed	3	17	38.95
Muscle-contraction	6	14	42.10
Total	18	62	

(Appendix A). Potential participants were required to have had a neurological screening including at least a CAT scan and/or a skull x-ray and/or an electroencephalogram, in order to rule out physical/organic causes of their headaches, prior to being accepted for participation in the project. A number of conditions that automatically excluded individuals from being potential participants in the study at this point were: a diagnosis of temporomandibular joint syndrome; structural or physical trauma (e.g. pinched nerves, concussion) associated with the onset of the headache history or the exacerbation of the headache symptoms; and sinus headaches that the individual could not distinguish from migraine or muscle-contraction headaches. Following the telephone contact, potential participants were scheduled for an interview for the purposes of determining a diagnosis of their headaches and formally including or excluding them from the project, based on the selection criteria described below. During this second, personal, interview session, potential participants signed a consent form (Appendix B) explaining the purposes and procedures of the diagnostic interview. Participants who met the inclusion criteria were requested to assist in the recruitment of a nonheadache control subject (friend or non-first-degree relative) of their same sex and approximate age (+/- 5 years). Matching on the variables of age and sex has been suggested by Blanchard and Andrasik (1982) following their

in-depth literature review on the problems apparent in headache research methodology. The headache participants' inclusion in the project, however, was not contingent on their success in procuring a control subject. The data from the first 20 nonheadache control subjects, who, as a group, did not significantly differ from the headache groups on the variables of age and sex and who successfully completed the assessment phase of the project (described below), were selected for inclusion in the data analyses. Headache participants were offered a minimum of 10 treatment sessions, at no cost to them, as remuneration for their participation in the project. This treatment was provided as part of another ongoing research project investigating treatment approaches for headaches.

Inclusion criteria. Each potential headache participant was independently interviewed and diagnosed by a doctoral student in clinical psychology using a structured headache intake form (Appendix C) and by a board-certified neurologist. Headache volunteers were included in the project if they received identical independent diagoses from the neurologist and the clinical psychology doctoral student. Those who received different diagnoses but for whom these discrepancies could be resolved, by a conference between the two diagnosticians, were also included.

The inclusion criteria for each group were based on the descriptions provided by the Ad Hoc Committee on

Classification of Headache (1962) and additional information provided by Diamond and Dalessio (1978), which are consistent with those used by previous researchers (e.g. Andrasik et al., 1982; Phillips & Hunter, 1981). The inclusion criteria were as follows:

1. migraine headache: Occurrence of at least two headaches per month which may or not be preceded by prodromata (e.g., visual or auditory experiences, paresthesias) and are characterized by at least three of the following: (a) usually unilateral onset; (b) described as throbbing or pulsating; (c) accompanied by nausea or vomiting, anorexia, constipation, or diarrhea; (d) accompanied by increased sensitivity to light, sounds, or odors; (e) a history of migraine in one or more first-degree relatives.

2. muscle-contraction headache: occurrence of an average of at least two headaches per week that are described as a dull, constant aching pain and/or as a tightness, cap- or band-like pressure on the head. In addition, individuals should report no more than one of the following vascular symptoms: unilateral pain, nausea, throbbing pain, visual prodromata.

3. mixed migraine and muscle-contraction headache: The individual must clearly identify that s/he has

two distinct types of headaches that meet the criteria for both migraine and muscle-contraction headaches as described above.

4. nonheadache controls: Controls were permitted to report experiencing an average of up to six headaches per year, none of which s/he considered problematic.

Measures

Headache monitoring record. The headache monitoring record (Appendix D) is a structured, nonstandardized form subjects complete on a daily basis. This recording form is a modified version of one previously used by Brantley (1980) and requires individuals to indicate, daily, whether or not they had a headache, the intensity of the headache on a Likert-type scale from 1 ("mild") to 4 ("severe"), and the duration of the headache (in hours). The headache record also requires respondents to provide information concerning other factors thought to be associated with headaches. This additional information was not used in the present study.

The Life Experiences Survey (LES). The LES (Appendix E) (Sarason et al., 1978) is a 57-item standardized, self-report measure that asks respondents to indicate which major life-events, of those listed, that they have experienced during the past 12 months. The first 47 items are common to individuals in the general

population and are very similar to items on the SRRS (Holmes & Rahe, 1967). The last 10 items are more specific to a student population and will not be used in the present study. The LES provides an index of the number of major life-events which have occurred and allows individuals to record their subjective ratings of the impacts of these events on a Likert-type scale from -3 ("extremely negative") to +3 ("extremely positive"). This rating scale will allow the separation of aversive events from pleasant events in contrast to the SRE or SRRS which lumps all events into one score. As previously mentioned in the literature review, it appears to be the more aversive events that correlate with negative affective states and symptoms. In addition, respondents indicate during which 6-month time period (i.e., the most recent or least recent during the previous 12 months) each event occurred (the SRE and SRRS do not allow this separation). Subsequently, the LES provides three scores; one for positive change, one for negative change, and a total change score for each 6-month period as well as for the entire 12-month period. Negative change scores from the LES have been found to correlate with anxiety and depression (Sarason et al., 1978), attitudes of mothers of at-risk infants (Crnic, Greenberg, Ragozin & Robinson, 1980), job satisfaction (Sarason & Johnson, 1979), academic achievement (Sarason et al., 1978), and menstrual discomfort (Siegel, Johnson & Sarason, 1979).

The Daily Stress Inventory (DSI). The DSI (Appendix F) (Brantley, Waggoner, Jones, & Rappaport, in press) is a 58-item standardized, self-report measure that assesses relatively minor daily stressful events, for example, arguments, social pressures, and job strains, that occur during a 24-hour time period. Respondents rate the stressfulness of each event they experienced on a Likert-type scale from 1 ("occurred but was not stressful") to 7 ("caused me to panic"). The inventory provides three daily stress scores: the number of stessful events that are experienced; the total sum of the ratings that are given to the items endorsed; and the average of the ratings that are given to the items endorsed. The items on this scale are different than those on the LES and tap less severe but potentially more frequently encountered stressful events (Brantley et al., in press). This scale has been found to have test-retest reliability coefficients in the low .60s, suggesting that it is more of a "state" measure and has the ability to fluctuate daily, and to significantly correlate with state anxiety and a monthly measure of daily stress (Brantley et al., in press). In addition, it has been found to discriminate between headache and nonheadache control subjects as well as between individuals producing abnormal compared to normal MMPI profiles (Brantley et al., 1984). The authors provide normative data for an adult sample.

The Multiple Affect Adjective Check List (MAACL).

The MAACL (Appendix G), "today" version (Zuckerman & Lubin, 1965), is a checklist of 132 adjectives. It measures day to day changes in three affects: anxiety, depression, and hostility. This scale has been found to have high internal consistency (.79 to .92). The test-retest reliability coefficients for the anxiety (.21), depression (.21), and hostility (.15) scales suggest that "this test is particularly suitable for the study of emotions ... where the measures may be expected to change markedly as a function of changing external or internal events" (Zuckerman, Lubin, Vogel, & Valerius, 1964, p. 423). Research with the MAACL has found it to be sensitive to experimentally induced affective states caused by disturbing movies, threat of examination, real examination, and threat of low grades (Zuckerman et al., 1964), and to hypnotically induced anxiety (Levitt, den Breeijen, & Persky, 1960) and hostility (Zuckerman et al., 1964). In addition, it has been found to be significantly related to stress in perceptual isolation (Zuckerman, Albright, Marks, & Miller, 1961), to stress in response to childbirth (Zuckerman, Gardiner, Vandiveer, Barrett, & den Breeijen, 1963), and to MAS scores (Zuckerman, 1960). Further, it has been shown to discriminate psychiatric patients from normals (Zuckerman, Lubin, & Robins, 1965) and to be a valid outcome measure of stress reduction following pharmacological treatment for anxiety and depression (Hankoff, Rudorfer, & Paley, 1962; Zuckerman et

al., 1965). Normative data is available for several normal and psychiatric samples.

Procedure

Following the diagnostic procedures, those headache subjects included in the project were scheduled to attend three 2-hour group assessment sessions, each separated by 2-week intervals. The headache participants were asked to bring their control participant, if they were successful in their recruitment of one, to the first and the two subsequent assessment sessions. The control sample was treated exactly the same as the headache participants throughout these three sessions.

During the first assessment session, the participants were assigned a project number and their written informed consent (Appendix H) was obtained. The project was explained to them in detail at that time and any questions they had were answered. In addition, participants completed a demographic information form (Appendix I) and were given instructions for completing the headache monitoring record, the DSI, and the MAACL. They were instructed to complete these forms daily, "starting today", throughout the entire 4 weeks of the project. Participants were instructed to complete these forms after 7:00 P.M. but before retiring each evening and were told that their responses should reflect the entire previous 24 hours. This response time-frame was provided in an effort to produce conformity in responding across subjects. As

part of the larger, ongoing project, the participants also completed a variety of other self-report psychological measures during this session. As they left this session, they were provided with enough daily forms for 14 days of recordings and given instructions to bring them back with them to the second assessment session to be held in two weeks.

During the second assessment session, all daily recording materials from the previous 2 weeks were collected and replaced with like forms for the second two weeks of recording. The forms from the first 2 weeks were inspected for completeness and to determine if the instructions were accurately followed. Any problems that were identified were discussed with the group and additional instructions were provided before the participants left this session. The participants completed another set of self-report psychological measures as part of the larger ongoing project. At the end of this session, they were instructed to return, with their daily recording forms, for the third and last assessment session in 2 weeks.

During the last assessment session, all daily recording materials were collected and inspected. Noncompliant participants, except control subjects, were, at this time, requested to complete an additional 2 weeks of daily forms before treatment would be provided. In addition, all participants completed the LES as the

instructions indicated. A final set of additional self-report psychological measures were also administered as part of the larger, ongoing project. At the end of this session, headache participants were assigned to a treatment group with the instructions that they would be contacted within the next 2 weeks.

As a result of the above procedure, headache and control subjects provided four consecutive weeks of daily information concerning their headache activity, the amount and number of daily stress(ors) they experienced, and their daily affective states, and scores from a major life-events measure.

Results

Data Reductions

The headache monitoring record. The number of headaches an individual experienced during the 4 weeks of monitoring were totaled across all days to provide the headache frequency score (HAF). The "1" to "4" intensity rating per headache provided the daily headache intensity score (HAI). Similarly, the duration (in hours) of each headache provided the daily headache duration score (HAD).

The MAACL. The three daily affect scores from the MAACL provided separate daily scores for anxiety (MAACL-ANX), depression (MAACL-DEP), and hostility (MAACL-HOS). In addition, each of the three daily scores were transformed into *t* scores, with a *M* = 50 and a *SD* = 10, and summed to provide an index to reflect

the intensity of an individual's daily total negative affective state (MAACL-INT). The transformation of these scores to standard scores was carried out in order to place the different means and standard deviations of the three separate MAACL scores on an equivalent scale (Cohen & Cohen, 1975). The mean and standard deviation of the total sample (N = 80) were used in obtaining these transformed scores.

The LES and DSI. Responses to the LES were reduced in two ways: according to the number of events endorsed and according to the subjective ratings that were given to the items endorsed. Scores reflecting the number of events endorsed included: (a) the number of events endorsed as having occurred during the previous 12 months and that were given a negative subjective rating by the respondent (TOTAL NEG NUM); (b) the number of events endorsed as having occurred during the least recent 6 months and that were given a negative subjective rating (NEG NUM FIRST); and (c) the number of events endorsed as having occurred during the most recent 6 months and that were given a negative subjective rating (NEG NUM LAST). Scores reflecting the amount of subjective stress experienced as a result of each item endorsed included: (a) the sum of the ratings of the negativley rated events that occurred during the entire previous 12 months (TOTAL NEG SUM); (b) the sum of the ratings of the negatively rated events that occurred during the least recent 6

months (NEG SUM FIRST); and (c) the sum of the ratings of the negatively rated events that occurred during the most recent 6 months (NEG SUM LAST).

Responses to the DSI provided two daily stress scores: the number of items endorsed (DSI-FREQ) and the sum of the impact ratings that were given to the endorsed items (DSI-SUM). The third score provided by the DSI, average impact rating, was not used in the present analyses because of its interdependency with the DSI-FREQ and DSI-SUM scores

Comparisons Between Groups on Age and Sex

Groups were compared with respect to their sex composition with a chi-square analysis. No differences between the four groups were found with regard to sex, $X^2(3, N = 80) = 4.87$, $p > .05$. Group differences with regard to age were examined with analysis of variance. The results of this analysis were not significant (see Appendix J). Thus, the control group does not significantly differ, with regard to age and sex composition, from the headache groups.

Correlations Among The Dependent Variables

A Pearson correlation matrix was constructed in order to guide the selection of the most appropriate procedures for the analyses of the data and to guide the selection of the variables to be included in these analyses. All of the scores on the dependent measures for the total sample ($N = 80$) were included in this analysis. The daily

scores from the headache monitoring record, except HAF, and from the DSI and the MAACL were averaged over the 28 days of the study. The results are presented in Table 2. The results of this analysis reveal that some of the measures are significantly, and highly, correlated with each other. This is particularly obvious when examining the correlations of scores derived from the same measurement instrument, for example, between the LES scores and between the MAACL scores, which often exceeded .80. As a result, the analyses that follow take the interrelatedness of these scores into consideration when possible. The only significant correlations between scores from different measurement instruments appeared between the MAACL scores and the LES scores. In general, these significant correlations suggest that the greater the number of negatively rated major life-events that individuals endorsed and the greater the negative impact of these events, the more anxious, depressed, and hostile individuals reported they were. However, the correlations between these scores are modest, sharing less than 10% common variance (see Table 2). Of the dependent measures used in the present study, only the DSI-FREQ score significantly correlated with any headache activity score. This significant correlation indicated, contrary to expectations, that as the number of daily stressors endorsed increased, the number of headaches reported decreased.

Table 2

Pearson Correlation Matrix Among Headache Activity, Stress, and Affect Measures for the Total Sample

	Headache Activity[a]			DSI[b]		MAACL[c]			
Measure	Freq	Int	Dur	FREQ	SUM	Anx	Dep	Hos	Int
Headache Activity									
Freq	-	.61**	.50**	-.25*	-.13	.10	.17	-.01	.09
Int		-	.73**	-.20	-.02	.11	.14	.05	.11
Dur			-	-.16	.01	.10	.15	.05	.11
DSI									
FREQ				-	.84**	-.04	-.12	.02	-.05
SUM					-	.19	.02	.05	.09
MAACL									
Anx						-	.88**	.71**	.92**
Dep							-	.82**	.97**
Hos								-	.91**

Table 2 (con't)

	LES NEG NUM[a]			LES NEG SUM*		
Measure	FIRST	LAST	TOTAL	FIRST	LAST	TOTAL
Headache						
Activity						
Freq	-.03	.03	.01	-.05	.06	.03
Int	-.04	.01	.02	-.03	.05	.02
Dur	-.05	-.05	-.07	-.04	-.02	-.03
DSI						
FREQ	.17	-.01	.10	.15	-.04	.05
SUM	.21	.11	.20	.19	.09	.17
MAACL						
Anx	.21	.23*	.31**	.20	.26*	.32**
Dep	.25*	.17	.28**	.23*	.18	.26*
Hos	.24*	.11	.23*	.18	.10	.18
Int	.25*	.18	.29**	.21	.19	.27**
LES NEG NUM						
LAST	-	.03	.61**	.95**	.02	.51**
FIRST		-	.82**	.05	.95**	.82**
TOTAL			-	.59**	.77**	.94**

Table 2 (con't)

LES NEG SUM			
LAST	-	.04	.56**
FIRST		-	.85**

Note. N = 80.

[a]Scores represent the number of headaches over 28 days (Freq), average of ratings of intensity from 1 ("mild") to 4 ("severe") per headache (Int), and average duration (in hours) per headache (Dur).

[b]Scores from the Daily Stress Inventory, averaged over 28 days.

[c]Anxiety (Anx), depression (Dep), and hostility (Hos) scores from the Multiple Affect Adjective Checlist, averaged over 28 days. These three scores were transformed to t scores and summed to provide a score representing total combined negative affect (Int).

[d]Scores from the Life Experiences Survey indicating the number of negatively rated events endorsed during the most recent 6-month time period (LAST), the least recent 6-month time period (FIRST), and the total of these two time periods (TOTAL).

[e]Scores from the Life Experiences Survey indicating the sum of the negative ratings given to the items endorsed for the most recent 6-month time period (LAST), the least recent 6-month time period (FIRST), and the total of these two time periods (TOTAL).

*$p < .05$. **$p < .01$.

Group Differences in Headache Activity

Because of the significant intercorrelations between the HAF, HAI, and HAD scores, group differences in headache activity were initially examined with multivariate analysis of variance (MANOVA). For this analysis, the HAI and HAD scores were averaged over the 28 days. The HAF score already reflects the total number of headaches over the 28 days. This overall analysis was significant, Wilks' $F(9, 180) = 11.51$, $p < .001$, indicating that the four groups differed on at least one of the indices of headache activity. In post-hoc analyses, each of the three dependent measures from the Headache Monitoring Record was compared across the four groups using univariate analysis of variance (ANOVA). The results indicate that the four groups significantly differed with respect to headache frequency, $F(3, 76) = 29.31$, $p < .001$, headache intensity, $F(3, 76) = 25.04$, $p < .001$, and headache duration, $F(3, 76) = 14.08$, $p < .001$ (see Appendix K). Table 3 presents the means and standard deviations for each group on each headache activity measure, along with the results of post-hoc Tukey's Studentized Range (HSD) tests when the initial F was significant. The post-hoc analyses revealed, as expected based on the selection criteria, that all three headache groups reported more headaches during the 4 weeks than did the control group and that the muscle-contraction group reported significantly more

headaches than both the mixed and migraine groups, which did not differ from each other (see Table 3). In addition, the headache groups described their headaches as being significantly more intense and of a longer duration than those of the control group. The headache groups did not, however, differ from each other on headache intensity or headache duration.

Group Differences in the Occurrence of Stressful Life-Events, Subjective Stress, and Affective Responses

Differences between the headache and control groups with respect to the occurrence of stressful life-events, subjective stress, and affective responses during the 28 days of the study were examined by MANOVA. A MANOVA procedure was used because, as shown in Table 2, many of the dependent measures are related to each other and this procedure accounts for possible correlations among the dependent measures while testing the set of variables simultaneously. The set of dependent variables used in this analysis was: (a) TOTAL NEG NUM, (b) NEG NUM FIRST, (c) NEG NUM LAST, (d) DSI-FREQ, (e) TOTAL NEG SUM, (f) NEG SUM FIRST, (g) NEG SUM LAST, (h) DSI-SUM, (i) MAACL-ANX, (j) MAACL-DEP, and (k) MAACL-HOS. The daily measures, variables d, h, i, j, and k, were averaged across the 28 days for this analysis. The overall MANOVA was not significant, Wilks' $F(27, 199) = .90$, $p > .05$.

The group means and standard deviations for each dependent

Table 3

Group Means on Headache Activity Parameters

	Group[a]			
	Control	Migraine	Mixed	Muscle-Contraction
Frequency[b]				
M	2.20$_c$	14.75$_b$	15.10$_b$	20.80$_a$
SD	4.07	6.89	6.58	7.79
Intensity[c]				
M	.66$_b$	2.21$_a$	2.01$_a$	2.03$_a$
SD	.79	.51	.54	.68
Duration[d]				
M	1.64$_b$	7.74$_a$	6.92$_a$	6.51$_a$
SD	2.45	4.20	2.86	3.38

Note. Means having different subscripts are significantly different at $p<.05$ using Tukey's HSD test.

[a]n = 20 for each group.

[b]Number of headaches over 28 days.

[c]Average intensity per headache rated from "1" (mild) to "4" (severe).

[d]Average, in hours, per headache.

variable are presented in Table 4.

As predicted, this analysis did not reveal significant group differences with respect to the number of negatively rated major life-events they report or with respect to the number of daily stressful events they report. Contrary to expectations, the headache groups also did not differ from the control group with respect to the intensity of their subjective reactions to major and minor stressful life-events or with respect to the level of anxiety, depression, and hostility they experienced during the 4 weeks of the study. Further, the means presented in Table 4 do not support the hypothesis that the headache groups consistently fall on a continuum of psychological distress with muscle-contraction headache sufferers at the most distressed end and migraineurs at the least distressed end. In fact, muscle-contraction headache sufferers did not score the highest, which indicates the most distress, on any of the dependent variables. In a secondary MANOVA, the variable MAACL-INT replaced the variables MAACL-ANX, MAACL-DEP, and MAACL-HOS. This variable was included to determine if the groups differed in the combined degree of anxiety, depression, and hostility they experienced, regardless of the specific type of affect. The three separate MAACL scores were excluded from this analysis because they are not independent of the MAACL-INT score. The result of this analysis was also nonsignificant, Wilks' $F(21,$

Table 4

Group Means and Standard Deviations on Stress and Affect Measures

	Group[a]			
Variable	Control	Migraine	Mixed	Muscle-Contraction
MAACL-ANX[b]				
M	7.78	8.65	9.01	8.67
SD	2.32	2.14	2.66	3.05
MAACL-DEP[b]				
M	15.80	17.81	18.72	17.65
SD	4.53	3.52	2.90	4.39
MAACL-HOS[b]				
M	9.67	10.17	11.11	10.04
SD	2.90	2.42	1.41	3.07
MAACL-INT[c]				
M	143.63	150.74	155.80	150.16
SD	20.25	16.36	14.26	23.04
DSI-FREQ[d]				
M	12.70	10.76	9.52	8.85
SD	8.08	6.86	4.15	5.80
DSI-SUM[d]				
M	28.72	29.53	23.89	22.76
SD	21.40	20.74	15.59	14.07

Table 4 (con't)

NEG NUM FIRST[a]				
M	1.20	1.85	1.35	1.30
SD	1.61	2.78	1.93	2.15
NEG NUM LAST[a]				
M	1.65	1.30	1.30	1.40
SD	1.79	1.53	1.26	1.67
TOTAL NEG NUM[a]				
M	2.85	3.15	2.65	2.70
SD	2.70	2.94	2.50	2.74
NEG SUM FIRST[b]				
M	2.10	3.50	2.60	2.95
SD	3.04	6.44	3.63	5.59
NEG SUM LAST[b]				
M	3.15	2.40	2.50	2.20
SD	3.75	2.46	2.98	2.84
TOTAL NEG SUM[c]				
M	5.25	5.90	5.10	5.15
SD	5.43	6.38	5.57	6.11

[a]n = 20 for each group.

[b]Average of 28 daily scores from the Multiple Affect Adjective Checklist.

[c]Average of daily MAACL scores transformed to t scores and summed.

[d]Average of 28 daily scores from the Daily Stress Inventory.

[e]Number of negatively rated events endorsed during 7-12 months on the Life Experiences Survey (LES).

[f]Number of negatively rated events endorsed during 0-6 months on the LES.

[g]Total number of negatively rated events from the LES.

[h]Sum of negative ratings of events for 7-12 month period on the LES.

[i]Sum of negative ratings of events for 0-6 month period on the LES.

[j]Total sum of negative ratings of events from the LES.

202) = .87, $p > .05$, indicating that there are no significant group differences. The group means for MAACL-INT are also included in Table 4. The hypothesis that headache groups differ in the degree of their combined daily affective states is not supported.

The Relations Among Headache Activity and the Occurrence of Stressful Life-events, Subjective Stress, and Affective Responses

The purpose of the following analyses was to determine if the headache groups differed with respect to how well the indices of stress and affective responses predicted headache activity. Stated another way the question was, "Is the relation between stress and/or affect and headache activity different across different headache diagnostic groups?" In order to answer this question, a series of multiple regression analyses, using group membership, 11 indices of stress and affective responses, and the interaction between the two as predictor variables, were conducted. Each of the 11 indices of stress and affective responses (variables "a" thru "k", described above), with the daily scores averaged over the 28 days, was analyzed in a separate multiple regression equation. Although this procedure produced a total of 33 regression analyses, the level of probability used to determine that each equation was significant was held constant, at $p < .05$, rather than adjusting for the family error rate. This was done for two reasons:

(a) because the present study is the first, in the headache literature, to investigate this question and a significant finding, even at this more liberal level, would warrant further research, and (b) the predictor of most interest in these equations was the interaction rather than the abilities of the individual overall models to predict headache activity. A significant interaction would indicate that the amount of variance in headache activity accounted for by the stress or affect score included in the analysis was affected by headache group membership. With regard to headache frequency, only the overall regression equations including MAACL-ANX, $F(5, 54) = 2.35, p < .05$, MAACL-DEP, $F(5, 54) = 2.63, p < .05$, and MAACL-HOS, $F(5, 54) = 2.74, p < .05$, as the stress and affect predictors were significant (see Appendix L). In these regressions, diagnosis was the only predictor that accounted for a significant amount of variance in headache frequency and none of the interactions between diagnosis and the stress and affect scores were significant. These findings indicated that the relations between stress and affect scores and headache frequency did not differ among the headache groups. None of the overall regression equations predicting headache intensity were significant (see Appendix M). These results indicated that the relations between stress and affect scores and headache intensity also did not differ between the headache groups.

Only the overall regression model using the variable NEG SUM LAST to predict headache duration was significant, $F(5, 54) = 2.75$, $p < .05$ (see Appendix N). In addition, the interaction between diagnosis and NEG SUM LAST was found to be significant in this regression equation, $F(2, 54) = 5.92$, $p < .05$. This significant interaction indicates that the relations between the subjective ratings of the stressfulness of major life-events occurring within the most recent 6-month period and the duration of headaches significantly differ among headache diagnostic groups. In order to investigate the nature of these differences, the correlations between headache duration and the NEG SUM LAST scores for each group, presented in Table 5, were examined. These correlations were transformed, using Fisher's r to z transformation table, and their differences computed (Cohen & Cohen, 1975). The results indicate that the difference between the r-to$z$ transformed scores for the migraine and mixed groups is significantly different, $z_{diff} = 2.34$, $p < .02$. No other differences were significant. This measure of stress is significantly more related to the duration of migraine headaches than it is to the duration of mixed headaches. For the migraine group, it appears that individuals who report experiencing a greater negative impact as a result of the major life-events they experienced during the most recent 6 months have longer lasting headaches than mixed

Table 5

Correlations Between Headache Duration and NEG SUM LAST[a] for Each Headache Group

	Headache Group		
	Migraine	Mixed	Muscle-contraction
r	.60**	-.11	.28

Note. *n* = 20 per group.

[a]Score from the Life Experiences Survey indicating the sum of the negative ratings given to the items endorsed as occuring during the most recent 6-month time period.

**$p < .01$.

headache sufferers.

In a secondary analysis, the variable MAACL-INT was entered into similar multiple regression analyses to determine if group differences exist in the relation between the overall degree of combined negative affect and headache activity. Only the overall regression model predicting headache frequency was significant, $F(5, 54) = 2.63$, $p < .05$, but no significant interaction effect was found (see Appendix L). Again, this finding suggests that the relations between this index of total negative affect and indices of headache activity do not differ among headache groups.

The Effectiveness of Combining Measures of Major and Minor Stressful Events in the Prediction of Headache Activity

Two sets of hierarchical stepwise multiple regression analyses were conducted to determine (a) whether scores of minor, daily stress will account for a significant amount of variance in headache activity after the variance accounted for by scores of stressful major life-events has been removed, and (b) whether or not scores of stressful major life-events will account for a significant amount of variance in headache activity after the variance accounted for by scores of minor, daily stressful events has been removed.

In the first set of analyses, three separate multiple regression analyses, one each with HAF,HAI, and HAD

serving as dependent variables, were conducted. The variables used to predict headache activity were TOTAL NEG NUM and TOTAL NEG SUM, measuring major stressful events, and DSI-FREQ and DSI-SUM, measuring minor stressful life events. The TOTAL NEG NUM and TOTAL NEG SUM scores were selected from among the LES scores because they reflect the total amount of major stress in an individual's life, including both of the 6-month time periods. The DSI-SUM scores were averaged across the 28 days of the study. The first step in these hierarchical regressions automatically included TOTAL NEG NUM and TOTAL NEG SUM in the equations and only allowed the DSI-FREQ and DSI-SUM scores to enter in subsequent steps if the increase in the variance accounted for in the dependent variable was significant at $p < .10$. The same predictor and criterion variables were also used in the second set of regressions, however, the DSI-FREQ and DSI-SUM scores were automatically entered in the first step while TOTAL NEG NUM and TOTAL NEG SUM were only allowed to enter in subsequent steps if the increase in the variance accounted for in the dependent variable was significant at $p < .10$. The .10 level of significance was chosen as the criterion in these analyses, rather than a more conservative level, because this is a new line of research and a signifcant finding at the .10 level would sill warrant further investigation in this area.

The results indicate that when the LES scores were

entered in the equations first, the DSI-FREQ score accounted for a significant amount of variance in headache frequency over and above the variance accounted for by the LES scores, however, the overall model was not significant, $F(3, 76) = 1.69$, $p > .05$ (see Appendix O). In addition, both DSI scores significantly entered the equations when the LES scores were entered first in the equation predicting headache intensity and the overall regression model was significant, $F(4, 75) = 2.89$, $p < .05$, accounting for 13.3% of the total variance in headache intensity scores (see Appendix P). Neither of the DSI scores significanly entered into the equation predicting headache duration and this overall model was not significant, $F(2, 77) = .46$, $p > .05$ (see Appendix Q).

When the DSI scores were entered in the equations first, none of the LES scores entered any of the equations. The overall regression models were significant for headache frequency, $F(2, 77) = 3.35$, $p < .05$, headache intensity, $F(2, 77) = 5.50$, $p < .01$, and headache duration, $F(2, 77) = 4.54$, $p < .01$ (see Appendices O, P, and Q). Although the multiple regression equations entering the DSI scores first are significant, the percent of variance in headache frequency, intensity, and duration they account for, 8.0%, 12.5%, and 10.5%, respectively, is modest at best.

These results suggest that measures of major

life-events do not account for significantly more variance in headache activity beyond the variance accounted for by measures of daily stress. On the other hand, measures of daily stress do account for a significant amount of variance in headache frequency and intensity beyond that accounted for by measures of major life-events. The amount of variance accounted for by combining the assessment of major and minor stress, however, is only significant when predicting headache intensity.

Group Differences in Affective and Subjective Reactions to Stress

The hypothesis that headache sufferers emotionally overreact to stress was examined with multivariate analysis of covariance (MANCOVA) procedures. The question of interest was, if the groups experience the same amount of stress, do they differ in the type or degree of their negative affective states? In order to answer this question, stress scores were statistically equated across the groups and the groups' stress ratings and affective responses were compared. A MANCOVA was used in which MAACL-ANX, MAACL-DEP, and MAACL-HOS served as dependent variables because they reflect the subjects' degree of anxiety, depression, and hostility, respectively. The MAACL scores were averaged across the 28 days for this analysis. Subjects' DSI-FREQ and DSI-SUM scores, averaged across 28 days, and their TOTAL NEG NUM and TOTAL NEG SUM scores were used as the covariates because they reflect

the amount of stress subjects reported experiencing.

The result of this MANCOVA was not significant, $F(6, 102) = .71$, $p > .05$. In a secondary MANCOVA the individual MAACL scores were replaced with the MAACL-INT score, all other variables remained the same. This analysis was also not significant, $F(2, 53) = 1.37$, $p > .05$. These results suggest that, with the number of stressors and the amount of subjectively experienced stress held constant, the groups do not differ in their combined degree of or type of affective reactions to stress. Thus, headache sufferers do not overreact, as compared to controls, to stress.

The Relation Between Headache Diagnosis and Indices of Stress and Affective Responses on Headache Versus Nonheadache days

According to the literature reviewed in the introduction, muscle-contraction headache sufferers should experience their greatest amount of stress on headache days as compared to nonheadache days occurring prior to their headaches and migraine sufferers should experience their greatest amount of stress on nonheadache days occurring prior to their headaches as compared to headache days. In order to investigate if these differences exist, the daily measures of stress and affect were separated into three groups of daily scores. Scores from nonheadache days two days prior to a headache were designated as "day 2" scores, scores from nonheadache days

one day prior to a headache were designated a "day 1" scores, and scores from days on which headaches occurred were designated as "headache day" scores. A 3 Diagnostic group X 3 Days MANOVA, with repeated measures across days, was performed with the following dependent variables averaged for each respective day: (a) DSI-FREQ; (b) DSI-SUM; (c) MAACL-ANX; (d) MAACL-DEP; and (e) MAACL-HOS. A MANOVA was used to allow for the simultaneous testing of indices of stress and affect while accounting for possible correlations among all of the dependent variables. This analysis allowed for the examination of the possible complex relation between stress indices and affective responses among Diagnostic groups, within groups across headache and nonheadache days, and the possible interaction between diagnosis and days. Of particular interest in this analysis are the Day and interaction effects. A significant main effect for days would indicate that headache subjects' scores on the dependent measures changed across days. A significant effect for the interaction would indicated that the different headache diagnostic groups' scores acted differently across headache and nonheadache days.

The results of this analysis revealed overall significant main effects for Days, Wilks' $F(10, 170) = 2.91$, $p < .01$, and for the interaction between Diagnosis and Days, Wilks' $F(20, 283) = 2.36$, $p < .01$, but no significant effect for Diagnosis, Wilks'

$F(10, 104) = .74$, $p > .05$. The nonsignificant results with regard to Diagnosis is not surprising given that the groups were not found to differ on these dependent measures in previous analyses. Post-hoc ANOVAs were performed on each dependent variable in order to examine which of the dependent measures contributed to the day and interaction effects. The results of these analyses indicated that the scores on all five of the dependent measures significantly changed across days at $p < .01$ for MAACL-ANX, MAACL-DEP, and DSI-SUM, and $p < .05$ for MAACL-HOS and DSI-FREQ (see Appendix R). The means and standard deviations of each variable for each group on each day are presented in Tables 6, 7, 8, 9, and 10. The different *ns* in these tables, for each group, are the result of some subjects not reporting any headache-free days or not reporting two consecutive headache-free days. Post-hoc Tukey's (HSD) tests revealed that the means for anxiety and depression were significantly higher on headache days than one day prior to a headache and that depression scores were also higher on headache days than two days prior to a headache (see Table 6). Post-hoc Tukey's tests did not reveal on which days the MAACL-HOS, DSI-FREQ, and DSI-SUM scores differed. In general, headache sufferers, regardless of diagnosis, are more anxious on headache days than they are one day prior to their headaches and more depressed on headache days than they are one and two days prior to their

Table 6

Mean Anxiety Scores for Headache Groups Across Headache and Nonheadache Days

Group	Day 2[a]	Day 1[b]	Headache
Migraine (n = 15)			
M	7.81	8.25	9.45
SD	3.18	2.89	2.04
Mixed (n = 18)			
M	8.57	8.41	9.39
SD	3.01	3.43	2.53
Muscle-contraction (n = 10)			
M	8.50	7.12	8.72
SD	2.80	2.87	3.14

Note. Anxiety scores are from the Multiple Affect Adjective Checklist. *M* = mean. *SD* = standard deviation.

[a]Scores from 2 days prior to a headache.

[b]Scores from 1 day prior to a headache.

Table 7

Mean Depression Scores for Headache Groups Across Headache and Nonheadache Days

Group	Day 2[a]	Day 1[b]	Headache
Migraine (n = 15)			
M	15.98	16.53	19.63
SD	5.25	4.94	3.53
Mixed (n = 18)			
M	17.74	18.35	19.21
SD	3.30	3.33	2.58
Muscle-contraction (n = 10)			
M	19.03	15.20	17.96
SD	3.41	5.44	4.59

Note. Depression scores are from the Multiple Affect Adjective Checklist. M = mean. SD = standard deviation.

[a]Scores from 2 days prior to a headache.

[b]Scores from 1 day prior to a headache.

Table 8

Mean Hostility Scores for Headache Groups Across Headache and Nonheadache Days

	Day		
Group	2[a]	1[b]	Headache
Migraine (n = 15)			
M	9.46	10.15	10.91
SD	3.14	2.97	2.81
Mixed (n = 18)			
M	10.53	10.93	11.41
SD	1.75	2.00	1.40
Muscle-contraction (n = 10)			
M	12.10	8.91	10.12
SD	4.25	3.76	3.24

Note. Hostility scores are from the Multiple Affect Adjective Checklist. M = mean. SD = standard deviation.

[a]Scores from 2 days prior to a headache.

[b]Scores from 1 day prior to a headache.

Table 9

Mean Number of Daily Stressors for Headache Groups Across Headache and Nonheadache Days

Group	Day 2[a]	Day 1[b]	Headache
Migraine (n = 15)			
M	11.67	12.13	11.19
SD	8.09	8.85	6.87
Mixed (n = 18)			
M	8.71	9.12	10.09
SD	3.94	4.86	4.75
Muscle-contraction (n = 10)			
M	7.86	8.32	9.14
SD	3.43	5.77	6.03

Note. The number of stressors is the frequency score from the Daily Stress Inventory. M = mean. SD = standard deviation.

[a]Scores from 2 days prior to a headache.

[b]Scores from 1 day prior to a headache.

Table 10

Mean Daily Subjective Stress for Headache Groups Across Headache and Nonheadache Days

Group	Day 2[a]	Day 1[b]	Headache
Migraine (n = 15)			
M	32.47	30.64	32.02
SD	29.74	25.35	20.20
Mixed (n = 18)			
M	21.09	23.03	25.87
SD	15.48	16.92	16.86
Muscle-contraction (n = 10)			
M	20.77	21.33	23.93
SD	12.73	13.55	14.92

Note. The subjective stress score is the SUM score from the Daily Stress Inventory. *M* = mean. *SD* = standard deviation.

[a]Scores from 2 days prior to a headache.

[b]Scores from 1 day prior to a headache.

headaches.

Post-hoc ANOVAs on the interaction effect revealed that the only significant interactions involved the MAACL-DEP scores, $p < .01$, and the MAACL-HOS scores, $p < .01$ (see Appendix R). The nature of these two interactions were investigated with Scheffe's comparisons which compared scores from pairs of days with pairs of Diagnostic groups. The comparisons between the migraine and muscle-contraction groups were of the most interest, as a result, one of these, the muscle-contraction group, was chosen as the group against which the other two groups were contrasted. Day 1 was chosen as the day against which the other two days were compared because it allowed the day-by-day progression of the scores, from day 2 to day 1 to headache day, to be examined. The results of these four comparisons for depression scores revealed that two of them exceeded the Scheffe critical difference value, $C_{crit} = 3.35$ at $p < .05$, that is needed to indicate that a significant interaction exists (see Appendix S). These comparisons revealed that the muscle-contraction group's, as compared to the migraine group's, scores were different from day 2 to day 1 and that the mixed group's, as compared to the muscle-contraction group's, scores also were different from day 2 to day 1. No significant interactions between any groups were found for depression scores from day 1 to the headache day. The nature of these significant

interactions are displayed in Figure 1. This figure shows that while migraine and mixed groups' scores increased from day 2 to day 1 the muscle-contraction group's scores decreased from day 2 to day 1. Similarly, the results of these four Scheffe's comparisons for hostility scores revealed that two of them exceeded the critical difference value, $C_{diff} = 2.92$ at $p < .05$, needed to indicate that a significant interaction exists (see Appendix T). Like the results using depression scores, the muscle-contraction group's hostility scores were different, as compared to both the mixed and migraine groups' scores, from day 2 to day 1 and no significant interactions were observed from day 1 to the headache day. Figure 2 illustrates that the musle-contraction group's scores decreased from day 2 to day 1 while the migraine and mixed groups' scores changed very little. In general, these results suggest that migraine and mixed headache sufferers become more depressed and hostile as their headaches approach as compared to muscle-contraction headache sufferers, who become less depressed and hostile the day before their headaches.

In a secondary MANOVA, the MAACL-ANX, MMACL-DEP, and MAACL-HOS scores were replaced with MAACL-INT scores to examine whether or not the groups differed with respect to the overall degree of their negative affective states across days irrespective to the particular type of affective response. DSI-FREQ and DSI-SUM scores were also

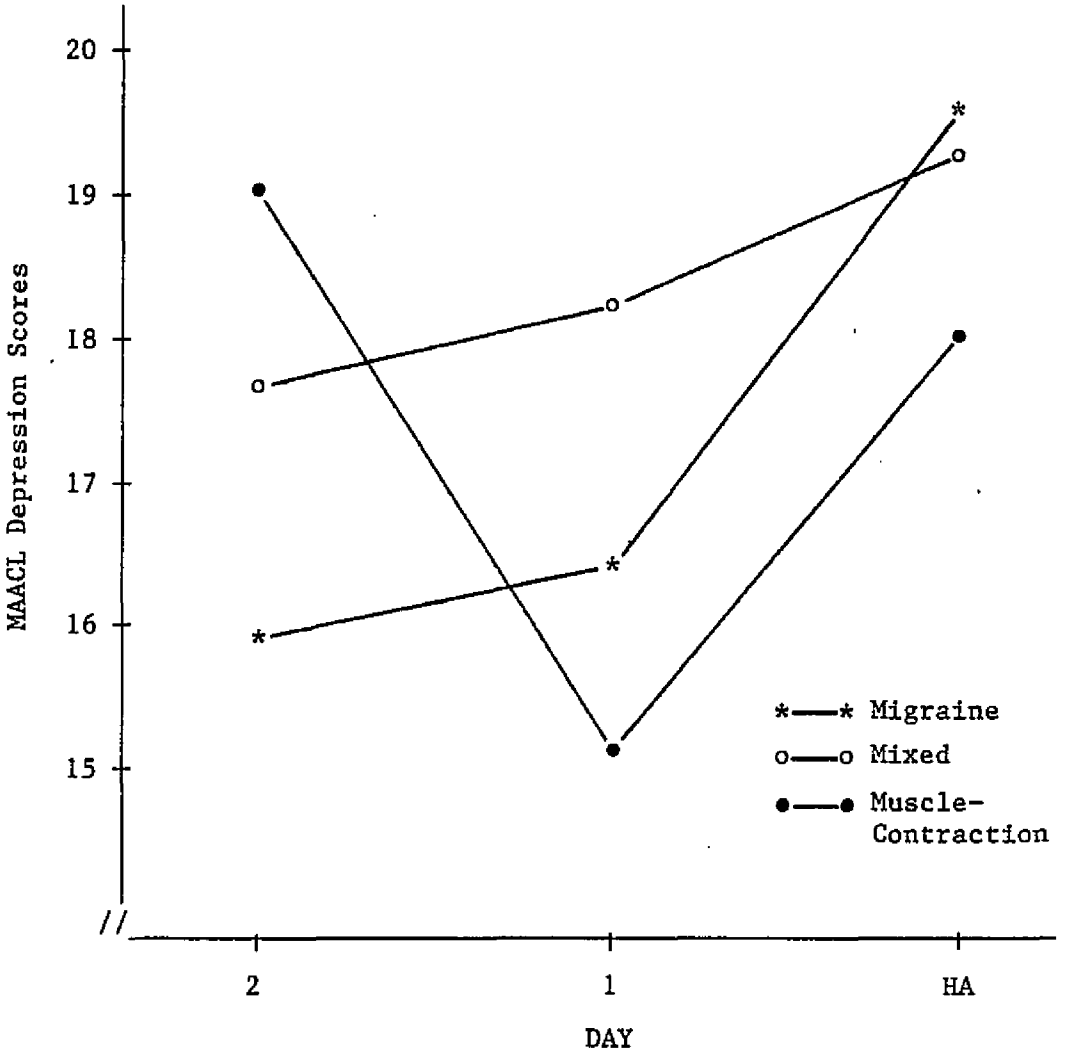

Figure 1. Headache groups' depression scores across pre-headache and headache days.

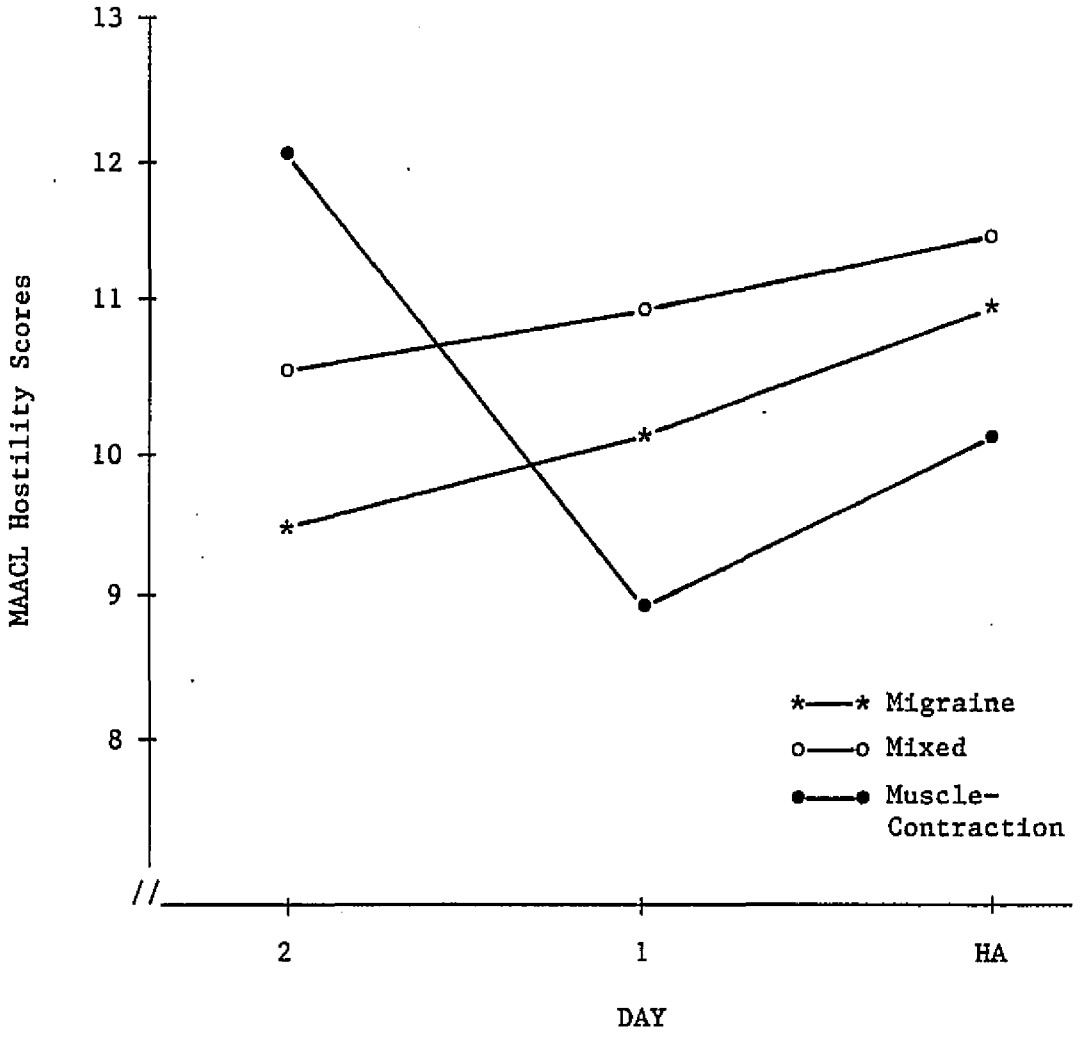

Figure 2. Headache groups' hostility scores across pre-headache and headache days.

included as dependent measures in this analysis. The results of this analysis, like those using the individual MAACL scores, revealed a significant main effect for Day, Wilks' $F(6, 174) = 3.47$, $p < .01$, and the interaction, Wilks' $F(12, 230) = 2.05$, $p < .05$, but no significant effect for Diagnosis, Wilks' $F(6, 108) = 1.08$, $p > .05$. A Post-hoc ANOVA showed that differences on the MAACL-INT score contributed to the main effect for Days, $p < .01$, and the effect for the interaction, $p < .05$ (see Appendix R). The means for each group on each day on the MAACL-INT score are presented in Table 11. Similar to the findings for the other MAACL scores, a post-hoc Tukey's test revealed that headache day scores were significantly greater than scores one day prior to a headache, regardless of headache group membership. Post-hoc Scheffe's comparisons, however, did not indicate which specific interactions between diagnoses and days were significant (see Appendix U).

Discussion

Do Headache Sufferers Lead Stressful Lives?

Even before the Ad Hoc Committee on Classification of Headache delineated the characteristics of 15 categories of headache in 1962, studies were presenting data indicating that life stress was an important contributing factor to both migraine and muscle-contraction headaches (Friedman et al., 1954; Selby & Lance, 1960). Since that

Table 11

Means of Intensity of Negative Affect for Headache Groups Across Headache and Nonheadache Days

	Day		
Group	2[a]	1[b]	Headache
Migraine (n = 15)			
M	143.52	147.40	157.98
SD	24.39	22.34	17.22
Mixed (n = 18)			
M	151.35	153.11	158.41
SD	16.78	18.17	12.95
Muscle-contraction (n = 10)			
M	157.65	138.92	151.05
SD	23.53	25.98	24.00

Note. Intensity of affect scores were derived from the total sum of the individual scores from the Multiple Affect Adjective Checklist after transformed to *t* scores. *M* = mean. *SD* = standard deviation.

[a]Scores from 2 days prior to a headache.

[b]Scores from 1 day prior to a headache.

time, numerous other survey studies, published as recently as 1983, have compiled a large body of data in support of this notion (e.g., Drummond, 1983; Featherstone & Beitman, 1983). Anyone would be hard pressed to contest these findings. When headache populations are compared to control populations with objective measures of stress such as used in the present study, however, no differences have emerged. There are a number of reasons for these apparently discrepant results. The most obvious difference between the studies concluding that headache sufferers lead stressful lives and those, including the present study, concluding that they have no more stress in their lives than a nonheadache control group is that none of the survey studies included control groups. No studies including a comparison population have found headache sufferers to experience more stress in their lives. Another obvious difference between these studies concerns the way in which stress was measured. All of the studies concluding that headache sufferers lead no more stressful lives than controls used standardized and objective measures of stress whereas the results of the survey studies were based on subjective, nonstandardized, and, often, unstructured procedures. It is argued here that these two methods of measuring stress may in fact be measuring different but overlapping facets of the construct called "stress". By standardizing and objectifying the measurement of stress with meaures like

the SRRS we may lose some aspects of the stress "experience" and come away with a less comprehensive picture. The present study attempted to increase the comprehensiveness of the assessment of stress by allowing subjective and idiographic ratings of stressful events and by assessing daily stressful events as well as major life-events. Inspite of this, the groups did not differ in the amount of stress they reported to have experienced. Although disappointing, it appears that it does not make any difference whether just the number of stressful events are counted or whether individuals' ratings of the stressfulness of the events are taken into account when assessing stressful life-events. Headache sufferers do not score differently than control subjects on these measures. This does not mean, however, that headache sufferers do not experience stress because they do report that stressul events occurred. The possibility remains that measures like the LES and DSI, when used alone, are not sensitive to differences between headache and control groups. The hypothesis most often offered, to explain why headache groups do not score differently than controls on measures of life events is that, headache sufferers do not experience more stess but, rather, "overreact" to the same amount of stress (Andrasik & Holroyd, 1980).

Do Headache Sufferers Emotionally Overreact to Stress?

The headache groups were not found to be more anxious, depressed, or hostile than controls. Although

most studies measuring psychopathology with personality inventories find at least one headache group, most often muscle-contraction groups, to significantly differ from a nonheadache control group (e.g., Anderson & Franks, 1981; Andrasik et al., 1982a; Kudrow & Sutkus, 1979), the present results are not necessarily contradictory for several reasons. The scores used from the MAACL are considered measures of affective states rather than of more enduring traits as measured by personality inventories. Therefore, the conclusion that the headache groups are no more anxious, depressed, or hostile than control subjects does not extend beyond the 28-day time frame assessed here. It is possible that differences could emerge with longer assessment periods. Another explanation for the lack of differences between groups could be that although the groups do not differ on daily scores averaged across time, these averaged scores mask group differences in day-to-day fluctuations of affective states.

No previous studies have assessed subjects' self-perceived emotional reactions to stressors. Instead, the subjects' ratings of the stressfulness of these stressors were used to indicate the subjects' reactions to the stressors. The viewpoint taken here is that the subjective ratings of stressfulness only verify that the subjects perceive the events as stressful and indicate the amount of stress individuals perceive themselves to have

been exposed to rather than indicate their emotional response to the event. This definition of stress is consistent with the interaction theories where stress is defined both in terms of the specific types of stressors an individual is exposed to (e.g., major or minor stressors) along with each subject's evaluation of the stressfulness of the stressor, which interact to produce an emotional response. Having described this theoretical viewpoint, it should be clear why the hypothesis that headache sufferers emotionally overreact to stress was examined, in this study, by statistically equating the groups on stress scores, including subjective ratings of stress, and measuring the degree of anxiety, depression, and hostility reported by the group members.

This variation in the definition of stress did not reveal group differences in the degree of or type of their affective responses thus failing to support the hypothesis that headache sufferers emotionally overreact to stress. Further, these results do not corroborate the notion that migraineurs are stimulus intensity "augmenters" who are more sensitive to stressors or experience stressors more intensely than muscle-contraction headache sufferers (Gannon et al., 1981; Klein, 1983). To conclude, however, that headache sufferers do not emotionally overreact, as compared to controls, to stressful life-events is premature for several reasons. First, several laboratory studies have found the subjective reactions of different

types of headache sufferers to differ from controls and from each other in response to standard stressors. Although the nature of and type of stressors used in laboratory studies are highly consistent across subjects, no such degree of control over the real-world stressors that subjects are exposed to seems possible short of attempting to match subjects on the specific major or minor events they endorse as having occurred. On the other hand, it may prove difficult to find an event or stressor that different individuals experience in the same way. According to interaction theories of stress, a host of mediational processes determine how stressful an individual perceives a stressor to be which, in turn, determines the extent of their emotional and physiological reactions to the stressor (Lazarus & Folkman, 1982). Consequently, differences in reactions to stressful life-events may be revealed only after these mediational processes, for example genetic make-up, past experience, personality traits, attitudes, and physiological states, are taken into consideration. In view of the present findings, it seems plausible that both person and environment variables may determine an individual's response to a stressful life-event (Lazarus, DeLongis, Folkman, & Gruen, 1985). As Laux and Vossel (1982) have pointed out, laboratory studies and field studies are both useful for studying the stress process, albeit to answer different questions. The need for more environmental

control may render the laboratory as the more appropriate environment for comparing emotional reactions to stressful events, leaving only the problem of controlling for the person variables.

A second reason why it is premature to conclude that headache sufferers do not overreact, as compared to control subjects, to stressful life-events based on the findings of the present study is that only one of several possible response systems was measured. Past research has demonstrated that stress can produce autonomic nervous system reactions, biochemical changes, and behavioral changes as well as self-perceived emotional reactions (see Baum, Grunberg, & Singer, 1982, for review). Headache and control groups have not been found to consistently differ in terms of the magnitude of their responses within one response system, for example in autonomic nervous system responses, as demonstrated in the numerous studies reviewed in the introduction, or in their affective responses, as in the present study. The suggestion has been made, however, that headache sufferers may differ in their patterns of responses across response systems. For example, Brantley (1980) found that his migraine group showed their greatest physiological responses to stimuli possessing an emotional component. In addition, Bakal (1975) has suggested, that after repeated exposure to stress, events may no longer be perceived as stressful and produce no emotional response but still be able to produce

the conditioned physiological changes necessary for the occurrence of a headache. These findings suggest that future research investigating differences in reactions to stressful events may find differences when several response systems are measured simultaneously.

Are Stress and Affect Related to Headache Activity?

This question was first addressed by the simplest method, correlations between each variable and indices of headache activity. Because a large number of measures were included in the correlation matrix (Table 2) the relations among the measures and headache activity will be discussed only in general terms. The most obvious finding that emerged from the correlation table is that when measures of stress and affect are taken individually, they are not significantly related to headache activity. Only one significant correlation, out of 46, emerged between the measures of affect and stress and headache activity. Surprisingly, this relation suggests that as the number of daily stressors increase, the number of headaches a subject experiences decrease, contrary to what is expected. This finding could indicate that someone who has many headaches tends to be less active and avoid stressful circumstances. Little importance is given to this finding because, out of 46 correlations, it could also have easily emerged due to chance. Also perplexing are the lack of any other significant correlations with headache activity. This suggests that headache activity

is not related to stress and affect, which is contrary to a number of studies suggesting that stress and anxiey and depression are all related to headache activity. Another possibility is that headache activity is related in some nonlinear fashion to stress and affect. Nonlinear stress-disorder relations have been suggested by stress thoerists. For example, some theorists have suggested that an individual must experience some minimum amount of stress before any risk of disorder arises (Crandall & Lehman, 1977; Holmes & Rahe, 1967; Lloyd, Alexander, Rice, & Greenfield, 1980). Hough, Fairbank, & Garcia (1976) have suggested that an asymptote may exist above which further stressful events do not appreciably increase the risk for disorder. A third possibility concerns the characteristics of the headache samples used in this study. Table 3 shows that, as a group, the headache subjects used in the present study reported experiencing a large number of headaches. On the average, the migraine and mixed headache sufferers reported experiencing a headache on one-half of the 28 days of monitoring and the muscle-contraction headache sufferers reported experiencing a headache on 21 out of 28 days. The frequencies of headaches reported by the subjects in the present study, however, are comparable to the frequencies in headache samples used by others (Blanchard et al., 1984; Demjen & Bakal, 1981; Henryk-Gutt & Rees, 1973). The problem that may have arisen with the use of headache

sufferers with frequent headaches is one of restricted range. The sampling procedure used in the present study may have inadvertently restricted the ranges of the headache indices to the point of lowering the correlations between them and the stress and affect scores to nonsignificant levels (Cohen & Cohen, 1975). If this is so, future studies should not require paricipants to report having a minimum number of headaches as an inclusion criterion.

A fourth possible reason for the lack of correlations between indices of headache activity and indices of stress and affect is that each variable, taken alone, does not account for a significant amount of variance in headache activity whereas if measures of stress and affect are combined with other factors, for example, biological, psychological, and social, significant relations with indices of headache activity could emerge (Blanchard et al., 1984). In fact, when the two indices of daily stress were entered into regression equations they accounted for a significant amount of variance in headache frequency, intensity, and duration.

Combining measures of major and minor stressful life-events for predicting headache activity. There appears to be merit in combining measures of stress associated with major and minor life-events for predicting headache activity. This is particularly the case for predicting headache intensity. Both of the daily stress

scores accounted for a significant amount of the variance in headache intensity that was not accounted for by the stress associated with major life-events. The reverse is not true, however. It seems that measures of the stress associated with major life-events do not account for a significant amount of the variance in headache frequency, intensity, or duration, above that already accounted for by daily stress. Further, none of the regression equations using only the major life-events scores significantly predicted headache activity whereas all three of the equations were significant when only the daily stress measures were included. These results lead to the conclusion that present headache activity is better predicted by concurrent measures of daily stress than by retrospective measures of major life-events. There is growing evidence that physical and psychological problems are more strongly related to daily, relatively minor stressful events than to major life-events. For example, DeLongis, Coyne, Dakof, Folkman, & Lazarus (1982) found their measure of daily "hassles" to account for more variance, than major life-events, in the predicton of health status. One likely explanation for these findings is that the occurrence of daily stressful events may also be affected by the occurrence of major life-events. Consequently, daily stress scores not only share some variance with major life-events scores when predicting headaches, but also account for some of the variance in

headache activity independently from major life-events scores. The direction of causality between stress and headaches, however, remains uncertain. The idea that having a headache could cause someone to engage in less activity, be more irritable, and seek out a physician seems just as plausible as the idea that daily stressful events cause headaches. Thus, the finding of a significant relatonship between daily stress and headaches may reflect some bidirectional relation. Future research attempting to predict headache activity should include measures of daily stress in their assessment batteries. The notion that headaches are stress-related disorders is supported by the present results but other factors are clearly involved because a large amount of the variance in headache activity remains unaccounted for by these variables.

Are Stress and Affect Differentially Related to Specific Types of Headaches?

The possibility that the relations between stress and affect and headache activity are different for different types of headaches was suggested by the findings of Blanchard et al. (1984) and Brantley (1980). The results of the regression equations, using the interaction between the stress and affect scores and diagnostic group membership, provide limited support for the possibility that the relations between stress and headache activity are different for different types of headache. They do

not, however, support the hypothesis that affect is more strongly related to the headache activity of one headache group over another. Only one of the 33 regression equations revealed a significant interaction effect. This finding suggests that the amount of subjective stress associated with major life-events occurring in the past significantly predicts the duration of migraine headaches, but not mixed or muscle contraction headaches. Although this could have appeared purely because of chance, it is very similar to the results obtained in a different laboratory with a different headache population (Blanchard et al., 1984). Blanchard and his associates found that their major life-events score also significantly predicted their headache index, created by combining headache intensity and duration scores, for migraineurs but not for mixed or muscle-contraction headache sufferers. These findings add support to the notion that the stress associated with major life events is more strongly related to migraine headaches than it is to mixed headaches, but not as compared to muscle-contraction headaches. The lack of findings for significant interactions between daily affect scores and daily stress scores and headache diagnosis means that these scores do not differentially predict the headache activity of one headache group over another.

How Are Stress and Affect Related to Headache Activity?

The results discussed to this point have examined mean differences between groups on measures of stress and affect and the strength of the relations between headache activity and measures of stress and affect. The use of daily measures of stress and affect also allowed the examination of patterns of fluctuations in these scores both, with respect to when headaches occur and with respect to differences between headache diagnostic groups.

The use of daily measures of stress and affect revealed significant differences in the changes in these scores across headache and nonheadache days. The analyses of these daily changes revealed that headache sufferers, regardless of diagnosis, are more depressed and anxious on headache days as compared to the day before their headaches. In addition, they are more depressed on headache days than they are two days before their headaches. These changes parallel the findings of Feuerstein et al. (1983) and Harvey and Hay (1983). These two studies, however, only included migraine groups. The present findings indicate, further, that this pattern of affective change also holds for mixed and muscle-contraction headache sufferers. This suggests that it may be head pain that causes these mood changes and not something more particular to the specific type of headache experienced, a conclusion that has been reached by others (e.g., Andrasik et al., 1982a; Dalessio, 1980; Philips, 1976). This finding may also explain why headache

subjects with frequent headaches look more disturbed on measures of personality and psychopathology (Drummond, 1983; Harper & Steger, 1978). While the results also indicate that daily stress scores and hostility scores significantly fluctuate across days, no particular patterns of changes emerged. This is puzzling considering that the significance levels of the overall models were all $p < .05$. A significant but unexpected finding appeared when changes in depression and hostility were compared across groups. Contrary to the findings of Harvey and Hay (1983), the migraine group did not show a positive mood change the day before a headache as compared to two days before a headache. Instead, the muscle-contraction group's mood improved, relative to the migraine and mixed headache groups, across these two days. Daily fluctuations in affective states could eaily be masked by trait measures of anxiety, depression, and hostility when daily measures of affective states are averaged over time. Although headache groups have not been found to consistently differ on mean levels of anxiety, depression, and hostility, they do score differently on a day-to-day basis. At the simplest level, these findings raise the possibility that the moods of muscle-contraction headache sufferers improve due to the rare occurrence, at least in this sample, of subjects going two consecutive days without a headache. At a more complex level, these results raise the possibility that,

like migraine headaches, the mechanisms operating to produce muscle-contraction headaches may be initiated sometime, possibly two days, prior to the day of a headache. Changes in biochemical and psyhophysiological activity levels might also occur in specific patterns prior to a muscle-contraction headache. Researchers have found that EMG activity typically increases in response to stressors presented in the laboratory and that epinephrine and norepinephrine may be released in response to emotional tension (Mathew et al., 1982). In addition, the early studies conducted by Ostfeld and associates (Ostfeld, 1962; Ostfeld et al., 1957) led them to suggest that an increase in norepinephrine was associated with feelings of tension and that this could lead to the vasoconstriction of nutrient arteries and contraction of associated muscles. But, no one has conducted research to examine how long it might take for these changes to occur before a headache is experienced. If a 2-day progression of frontalis EMG activity was observed prior to the headache, where, for example, EMG activity increased two days before the headache, it could explain the discrepancies in the findings of research as to whether or not EMG activity increases on headache days as compared to nonheadache days. In speculation, a finding of no difference may be due to the inadvertent measurement of this high nonheadache day EMG. The finding that the migraine group's moods did not improve the day before a

headache as compared to two days before a headache does not necessarily detract from the hypothesis that the mechanisms that lead to them are initiated several days prior to the headache. The findings of Feuerstein et al. (1983) suggest that these mechanisms may be initiated up to 4 days preceding an attack. They found that while anxiety scores on headache days were significantly greater than 1, 2, and 3 days prior to the migraine, the anxiety on headache days was not significantly greater than anxiety scores 4 days prior to the migraine attack. Unfortunately, the headache sufferers in the present study had such frequent headaches that there were not enough headache-free days to allow this analysis. In general, these findings provide evidence suggesting that changes in affective states are associated with the occurrence of migraine, mixed, and muscle-contraction headaches and that the patterns of these changes in moods differ for the different types of headaches as the headache bout approaches.

Expanding the present methodology to include not only concurrent measures of daily stress and daily affect but also concurrent daily measures of, for example, psychophysiological activity, dietary changes, biochemical changes, and hormonal changes, might reveal complex interrelations that enhance our ability to predict when and to state why a particular headache occurs. Taken a step futher, significant results could have implications

for treatment by providing data to suggest choosing the use of one treatment strategy, for example, among antidepressants, biofeedback, dietary restrictions, and vasodilating drugs, over another.

In summary, the major findings of this study are that: (a) headache sufferers do not report leading more stressful lives than a control population; (b) headache sufferers do not, as compared to controls, emotionally overreact to life stress; (c) headache activity, regardless of diagnosis, is related, although only moderately so, to life stress; (d) measures of daily stress significantly predict headache activity and account for a significant amount of the variance in headache activity beyond that accounted for by measures of major life-stress alone; (e) measures of major life-stress do not account for a significant amount of the variance in headache activity beyond that accounted for by measures of daily stress; (f) different headache groups do not experience significantly different levels of negative affect during a 4-week monitoring period; (g) changes in daily stress levels are not related to the occurrence of a particular headache attack; and (h) changes in daily levels of depression and hostility, in relation to the occurrence of a headache, significantly differ across headache diagnostic groups.

Although conclusions, based on the above findings, cannot be taken beyond the confines of the characteristics

of the particular experimental groups used and the particular measures employed in this study, the differences obtained warrant further investigation. All of the significant findings were found by using a methodology that departed from the more traditional laboratory and life-events approaches to studying the stress-headache relation. The seemingly complex relations among stress, emotional states, and headache activity might be further delineated if future researchers include the concurrent assessment of daily stress levels, daily affective states, and headache activity in their methodologies. Focusing on the issues of whether or not headache groups experience more stess, more psychological disturbance, or overreact to stress, as compared to controls, has not been fruitful. The present results suggest that a more promising line of research is one focusing on <u>how</u> daily changes in stress levels and emotional states correspond with the occurrence of a headache.

References

Ad Hoc Committee on Classification of Headache (1962). Classification of headache. Journal of the American Medical Association, 179, 717-718.

Adams, H. E., Feuerstein, M., & Fowler, J. L. (1980). Migraine headache: Review of parameters, etiology, and intervention. Psychological Bulletin, 87, 217-237.

Alexander, F. (1950). Psychosomatic medicine: Its principles and applications. New York: W. W. Norton.

Alvarez, W. C. (1974). The migrainous personality and constitution: The essential features of the disease: A study of 500 cases. American Journal of Medical Science, 2, 213.

American Psychiatric Association (1980). Diagnostic and statistical manual of mental disorders (3rd Ed.). Washington, DC: Author.

Anderson, C. D., & Franks, R. D. (1981). Migraine and tension headache: Is there a physiological difference? Headache, 21, 63-71.

Andrasik, F., Blanchard, E. B., Arena, J. G., Teders, S. J., & Rodichok, L. D. (1982a). A cross-validation of the Kudrow-Sutkus classification system for diagnosing headache type. Headache, 22, 2-5.

Andrasik, F., Blanchard, E. B., Arena, J. G., Teders, S.

J., Teevan, R. C., & Rodichok, L. D. (1982b). Psychological functioning in headache sufferers. Psychosomatic Medicine, 44, 171-182.

Andrasik, F., & Holroyd, K. A. (1980). Physiologic and self-report differences between tension and non-tension headache sufferers. Journal of Behavioral Assessment, 2, 135-141.

Anthony, M., Hinterberger, H., & Lance, J. W. (1967). Plasma serotonin in migraine and stress. Archives of Neurology, 16, 544-552.

Appenzeller, O. (1969). Vasomotor function in migraine. Headache, 9, 147-155.

Appenzeller, O. (1976). Monoamines, headaches, and behavior (pp. 43-48). In O. Appenzeller (ED.), Pathogenesis and treatment of headache (2nd Ed.). New York: Spectrum Publications.

Appenzeller, O., Feldman, R. G., & Friedman, A. P., (1979). Migraine, headache, and related conditions: Panel 7. Archives of Neurology, 36, 784-805.

Bain, S. T., & Spaulding, W. B. (1967). The importance of coding presenting symptoms. Canadian Medical Association Journal, 97, 953-959.

Bakal, D. A. (1975). Headache: A psychobiological perspective. Psychological Bulletin, 82, 369-382.

Bakal, D. A., Demjen, S., & Kaganov, J. A. (1981). Cognitive behavioral treatment of chronic headache.

Headache, 21, 81-86.

Bakal, D. A., & Kaganov, J. A. (1979). Symptom characteristics of chronic and nonchronic headache sufferers. Headache, 19, 285-289.

Bakal, D. A., & Kaganov, J. A. (1977). Muscle-contraction and migraine headache: A psychophysiological comparison. Headache, 17, 208-215.

Baum, A., Grunberg, N. E., & Singer, S. (1982). The use of psychological and neuroendocrinological measurements in the study of stress. Health Psychology, 1, 217-236.

Beaty, E. T., & Haynes, S. N. (1979). Behavioral intervention with muscle-contraction headache: A review. Psychosomatic Medicine, 41, 165-180.

Bihldorf, J. P., King, S. H., & Parnes, L. R. (1971). Psychological factors in headache. Headache, 82, 369-381.

Blanchard, E. B., & Andrasik, F. (1982). Psychological assessment and treatment of headache: Recent developments and emerging issues. Journal of Consulting and Clinical Psychology, 50, 859-879.

Blanchard, E. B., Andrasik, F., Arena, J. G., Neff, D. F., Jurish, S. E., Teders, S. J., Saunders, N. L., Pallmeyer, T. P., Dudek, B. C., & Rodichok, L. D. (1984). A bio-psycho-social investigation of headache activity in a chronic headache population. Headache, 24, 79-87.

Blau, J. N. (1971). Migraine research. British Medical Journal, 2, 751-754.

Botney, M. (1981). An inquiry into the genesis of migraine headache. Headache, 21, 179-185.

Brantley, P. J. (1980). Headache: The role of environmental and emotional stress. Unpublished doctoral dissertation, University of Georgia, Athens, GA.

Brantley, P., Waggoner, C., Jones, N., & Rappaport, N. (in press). The Daily Stress Inventory: Development, reliability, and validity. Journal of Behavioral Medicine.

Brown, G. W. (1981). Life events, psychiatric disorders and physical illness. Journal of Psychosomatic Research, 25, 461-473.

Burnstock, G. (1981). Pathophysiology of migraine: A new hypothesis. Lancet, 7, 1397-1399.

Callaghan, N. (1968). The migraine syndrome in pregnancy. Neurology, 18, 197-201.

Carroll, J. D. (1971). Migraine: General management. British Medical Journal, 2, 756-757.

Cinciripini, P. M., Williamson, D. A., & Epstein, L. H. (1980). Behavioral treatment of migraine headaches. In J. M. Ferguson & C. B. Taylor (Eds.), The comprehensive handbook of behavioral medicine: Syndromes and special areas (vol. 2) (pp. 207-227). New York: Spectrum.

Cleary, P. J. (1980). A checlist for life event research. Journal of Psychosomatic Research, 24, 199-207.

Cohen, J., & Cohen, P. (1975). Applied multiple regression/correlation analysis for the behavioral sciences. New York: John Wiley.

Cohen, M. J., Rickles, W. H., & McArthur, D. L. (1978). Evidence for physiological response stereotypy in migraine headache. Psychosomatic Medicine, 40, 344-354.

Cohen, R. A., Williamson, D. A., Monguillot, J. E., Hutchinson, P. C., Gottlieb, J., & Waters, W. F. (1983). Physiological response patterns in vascular and muscle-contraction headaches. Journal of Behavioral Medicine, 6, 93-107.

Crandall, J. E., & Lehman, R. E. (1977). Relationship of stressful life events to social interest, locus of control, and psychological adjustment. Journal of Consulting and Clinical Psychology, 45, 1208.

Crnic, K. A., Greenberg, M. T., Ragozin, A. S., & Robinson, N. M. (1980, March). The effects of life stress and social support on the life satisfaction and attitudes of mothers of newborn normal and at-risk infants. Paper presented at the Western Psychological Association Convention, Honolulu, HI.

Dalessio, D. J. (Ed.). (1980). Wolff's headache and other head pain(4th ed.). New York: Oxford

University Press.

Dalessio, D. J. (1978). Mechanisms of headache. Medical Clinics of North America, 62, 430-442.

Dalsgaard-Nielsen, T. (1965). Migraine and heredity. Acta Neurologica Scandinavia, 41, 287-300.

DeLongis, A., Coyne, J. C., Dakof, G., Folkman, S., & Lazarus, R. S. (1982). Relationship of daily hassles, uplifts, and major life events to health status. Health Psychology, 1, 119-136.

DeLongis, A., Coyne, J. C., Folkman, S., & Lazarus, R. S. (1982). Relationship of daily hassles, uplifts, and major life events to health status. Health Psychology, 1, 119-136.

DeLozier, J. E., & Gagnon, R. O. (1975). National Ambulatory Care Survey: 1973 summary, United States (DHEW Publications No. HRA 76-1772). Washington, DC: U.S. Government Printing Office.

Demjen, S. & Bakal, D. (1981). Illness behavior and chronic headache. Pain, 10, 221-229.

Derogatis, L. R. (1982). Self-report measures of stress. In L. Goldberger & S. Breznitz (Eds.), Handbook of stress: Theoretical and clinical aspects (pp. 270-294). New York: The Free Press, MacMillan.

Diamond, S. & Dalessio, D. J. (1978). The practicing physicians approach to headache (2nd ed.). Baltimore, MD: Williams & Wilkins.

Dohrenwend, B. S. & Dohrenwend, B. P. (1978). Some

issues in research on stressful life events. Journal of Nervous and Mental Disease, 106, 7-15.

Drummond, P. D. (1983). Predisposing, precipitating and relieving factors in different categories of headache. Headache, 23, 16-22.

Drummond, P. D., & Lance, J. W. (1984). Clinical diagnosis of headache symptoms. Journal of Neurology, Neurosurgery and Psychiatry, 47, 128-133.

Dunbar, F. (1954). Emotions and bodily changes. New York: Columbia University Press.

Engel, G. L., Ferris, E. B., & Romano, J. (1945). Focal electroencephalographic changes during the scotomatas of migraine. American Journal of Medical Science, 209, 650.

Epstein, L. H., Abel, G. G., Collins, F., Parker, C., & Cinciripini, P. M. (1978). The relationship between frontalis muscle activity and self-reports of head pain. Behavior Research and Therapy, 16, 153-160.

Epstein, L. H., & Cinciripini, P. M. (1981). Behavioral control of tension headache. In J. M. Ferguson and C. B. Taylor (Eds.), The comprehensive handbook of behavioral medicine: Syndromes and special areas (vol. 2) (pp. 229-240). New York: Spectrum.

Epstein, M. T., Hockaday, J. M., & Hockaday, T. D.

(1975). Migraine and reproductive hormones throughout the menstrual cycle. Lancet, 1, 543-547.

Featherstone, H. J., & Beitman, B. D. (1983). "Daily" common migraine: Psychosocial predictors of outcomes of medical therapy. Headache, 23, 110-112.

Feuerstein, M., Bortolussi, L., Houle, M., & Labbe, E. (1983). Stress, temporal artery activity, and pain in migraine headache: A prospective analysis. Headache, 23, 296-304.

Feuerstein, M., Bush, C., & Corbisiero, R. (1982). Stress and chronic headache: A psychophysiological analysis of mechanisms. Journal of Psychosomatic Research, 26, 319-324.

Friedman, A. P. (1979). Characteristics of tension headache: A profile of 1,420 cases. Psychosomatics, 20, 451-461.

Friedman, A. P. (1978). Migraine. Medical Clinics of North America, 62, 481-494.

Friedman, A. P. (1964). Reflection on the problem of headache. Journal of the American Medical Association, 190, 121-123.

Friedman, A. P. (1963). Studies in pharmacotherapy of headache. Neurology, 13, 27-33.

Friedman, A. P., von Storch, T. J., & Merritt, H. H. (1954). Migraine and tension headaches: A clinical study of two thousand cases. Neurology, 4,

773-788.

Fromm-Reichmann, F. (1937). Contributions to the psychogenesis of migraine. Psychoanalytic Review, 24, 26.

Gannon, L. R., Haynes, S. N., Safranek, R., & Hamilton, J. (1981). A psychophysiological investigation of muscle-contraction and migraine headache. Journal of Psychosomatic Research, 25, 271-280.

Goodell, H. (1967). Thirty years of headache research in the laboratory of the labe. Headache, 7, 158-171.

Haber, J. D., Kuczmierczyk, A. R., & Adams, H. E. (1985). Tension headaches: Muscle overactivity or psychogenic pain. Headache, 25, 23-29.

Hankoff, L. D., Rudorfer, L., & Paley, H. M. (1962). A reference study of ataraxics. A two week double blind outpatient evaluation. Journal of New Drugs, 2, 173-178.

Harper, R. G., & Steger, J. C. (1978). Psychological correlates of frontalis EMG and pain in tension headache. Headache, 18, 215-218.

Harrison, R. (1975). Psychological testing in headache: A review. Headache, 15, 177-185.

Hart, J. D., & Chichanski, K. A. (1981). A comparison of frontal EMG biofeedback and neck EMG biofeedback in the treatment of muscle-contraction headache. Biofeedback and Self-Regulation, 6, 63-74.

Harvey, P. G., & Hay, K. M. (1984). Mood and migraine - a preliminary prospective study. Headache, 24, 225-228.

Hawkins, N. G., Davies, R., & Holmes, T. H. (1957). Evidence of psychosocial factors in the development of pulmonary tuberculosis. American Review of Tuberculosis and Pulmonary Disease, 75, 768-780.

Haynes, S. N., Gannon, L. R., Cuevas, J., Heiser, P., Hamilton, J., & Kafranides, M. (1983). The psychophysiological assessment of muscle-contraction headache subjects during headache and nonheadache conditions. Psychophysiology, 20, 393-399.

Henryk-Gutt, R. & Rees, W. L. (1973). Psychological aspects of migraine. Journal of Psychosomatic Research, 17, 141-153.

Holmes, T. H., & Rahe, R. H. (1967). The Social Readjustment Rating Scale. Journal of Psychosomatic Research, 11, 213-218.

Holroyd, K. A., Andrasik, F., & Westbrook, T. (1977). Cognitive control of tension headache. Cognitive Therapy and Research, 1, 121-133.

Horton, B. (1956). Histaminic cephalgia: Differential diagnosis and treatment. Procedings of the Mayo Clinic, 31, 325.

Hough, R. L., Fairbank, D. T., & Garcia, A. M. (1976). Problems in the ratio measurement of life stress. Journal of Health and Social Behavior, 17,

70-82.

Howarth, E. (1965). Headache, personality and stress. British Journal of Psychiatry, 119, 1193.

Johnson, E. S. (1978). A basis for migraine therapy - the autonomic theory reappraised. Graduate Medical Journal, 54, 231-242.

Kanner, A. D., Coyne, J. C., Schaefer, C., & Lazarus, R. S. (1981). Comparison of two modes of stress measurement: Daily hassles and uplifts versus major life-events. Journal of Behavioral Medicine, 4, 1-39.

Klein, S. H. (1983). Perception of stimulus intensity by migraine and non-migraine subjects. Headache, 23, 158-161.

Kudrow, L. (1978). Current aspects of migraine headache. Psychosomatics, 19, 48-57.

Kudrow, L. (1976). Tension headache. In O. Appenzeller (Ed.), Pathogenesis and treatment of headache (pp. 81-91). Spectrum: New York.

Kudrow, L. (1975). The relationship of headache frequency to hormone use in migraine. Headache, 15, 36-40.

Kudrow, L., & Sutkus, B. J. (1979). MMPI pattern specificity in primary headache disorders. Headache, 19, 18-24.

Kunkle, E. C. (1959). Acetylcholine in the mechanism of headache of the migraine type. Archives of Neurology and Psychiatry, 81, 135.

Lacey, J. I., & Lacey, B. C. (1958). Verification and extension of the principle of autonomic response stereotypy. American Journal of Psychiatry, 71, 50-73.

Lance, J. W., & Anthony, M. (1966). Some clinical aspects of migraine: A prospective survey of 500 patients. Archives of Neurology, 15, 356-361.

Laux, L., & Vossel, G. (1982). Paradigms in stress research: Laboratory versus field and traits versus processes. In L. Goldberger & S. Breznitz (Eds.), Handbook of stress: Theoretical and clinical aspects (pp. 203-211). New York: The Free Press, MacMillan.

Lazarus, R. S. (1966). Psychological stress and the coping process. New York: McGraw-Hill.

Lazarus, R. S., DeLongis, A., Folkman, S., and Gruen, R. (1985). Stress and adaptational outcomes: The problem of confounded measures. American Psychologist, 40, 770-779.

Lazarus, R. S., & Folkman, S. (1982). Coping and adaptation. In W. D. Gentry (Ed.), The handbook of behavioral medicine (pp. 282-325). New York: Guilford Press.

Leviton, A. (1978). Epidemiology of headache. In Advances in Neurology (vol. 19) (pp. 43-50). New York: Raven Press.

Levitt, E. E., den Breeijen, A., & Persky, H. (1960).

The induction of clinical anxiety by means of a standardized hypnotic technique. American Journal of Clinical Hypnosis, 24, 457-462.

Lipowski, Z. J. (1977). Psychosomatic medicine in the seventies: An overview. The American Journal of Psychiatry, 134, 233-244.

Lloyd, C., Alexander, A. A., Rice, D. G., & Greenfield, N. S. (1980). Life events as predictors of academic performance. Journal of Human Stress, 6, 15-25.

Lucas, R. M. (1977). Migraine in twins. Journal of Psychosomatic Research, 20, 147-156.

Malmo, R., & Shagass, C. (1949). Physiologic study of symptom mechanisms in psychiatric patients under stress. Psychosomatic Medicine, 11, 25-29.

Malmo, R. B., & Smith, A. A. (1955). Forehead tension and motor irregularities in psychoneurotic patients under stress. Journal of Personality, 23, 391-406.

Martin, M. J. (1983). Muscle-contraction (tension) headache. Psychosomatics, 24, 319-324.

Martin, M. J. (1972). Muscle-contraction headache. Psychosomatics, 13, 19-26.

Martin, M. J. (1966). Tension headache, a psychiatric study. Headache, 6, 47-54.

Martin, P. R., & Mathews, A. M. (1978). Tension headaches: Psychophysiological investigation and treatment.

Journal of Psychosomatic Research, 22, 389-399.

Martin, M. J., Rome, H. P., & Swenson, W. M. (1967). Muscle contraction headache: A psychiatric review. Research Clinical Studies in Headache, 1, 184-204.

Masland, W. S. (1978). Electroencephalography and electromyography in the diagnosis of headache. Medical Clinics of North America, 62, 571-584.

Mathew, R. G., Weinman, M. L., & Largen, J. W. (1982). Sympathetic-adrenomedullary activation and migraine. Headache, 22, 13-19.

Maxwell, H. (1966). Migraine: Background and treatment. Baltimore, MD: J. Wright, Bristol.

McAnulty, D. P., Rappaport, N. B., Brantley, P. J., Waggoner, C. D., & Williamson, D. A. (1984, November). Psychological correlates of head pain. Paper presented at the annual meeting of The Association for the Advancement of Behavior Therapy, Philadelphia, PA.

Moskowitz, M. A., Reinhard, J. F., Romero, J., Melamed, E., & Pettibone, D. J. (1979). Neurotransmitters and the fifth cranial nerve: Is there a relation to the headache phase of migraine? Lancet, 5, 883-885.

O'Brien, M. D. (1971). Cerebral blood flow changes in the migraine headache. Headache, 11, 139-143.

Onel, Y., Friedman, A. P., & Grossman, J. (1961). Muscle blood flow studies in muscle-contraction headaches. Neurology, 11, 935-939.

Ostfeld, A. M. (1962). The common headache syndromes: Biochemistry, pathophysiology, therapy. Illinois: C. C. Thomas.

Ostfeld, A. M., Reis, D. J., & Wolff, H. G. (1957). Studies on headache: Bulbar conjunctival ischemia and muscle-contraction headache. Archives of Neurology and Psychiatry, 77, 113-119.

Parnell, P., & Cooperstock, R. (1979). Tranquilizers and mood elevators in the treatment of migraine: An analysis of the Migraine Foundation Questionnaire. Headache, 19, 78-86.

Passchier, J., van der Helm-Hylkema, H., & Orlebeke, J. F. (1984). Psychophysiological characteristics of migraine and tension headache patients: Differential effects of sex and pain state. Headache, 24, 131-139.

Pearlin, L. I. (1982). The social contexts of stress. In L. Goldberger & S. Breznitz (Eds.), Handbook of Stress: Theoretical and clinical aspects (pp. 367-379). New York: The Free Press, MacMillan.

Perkins, D. V. (1982). The assessment of stress using life events scales. In L. Goldberger & S. Breznitz (Eds.), Handbook of stress: Theoretical and clinical aspects (pp. 320-331). New York: The Free Press, MacMillan.

Philips, C. (1978). Tension headache: Theoretical problems. Behavior Research and Therapy, 16, 249-261.

Philips, C. (1977). The modification of tension headache pain using EMG biofeedback. Behavior Research and Therapy, 15, 119-129.

Philips, C. (1976). Headache and personality. Journal of Psychosomatic Research, 20, 535-542.

Philips, C., & Hunter, M. (1981). Pain behavior in headache sufferers. Behavioral Analysis and Modification, 4, 257-266.

Pozniak-Patewicz, E. (1976). Cephalic spasm of head and neck muscles. Headache, 15, 261-266.

Pratt, J. M., Williamson, D. A., Cohen, R. A., Granberry, S. W., & Jarrel, P. (1982). Psychological differences among headache populations: Stress, depression, anxiety and locus of control. Unpublished manuscript, Louisiana State University, Baton Rouge, LA.

Price, K., & Blackwell, S. (1980). Trait levels of anxiety and psychological responses to stress in migraineurs and normal controls. Journal of Clinical Psychology, 36, 658-660.

Rabkin, J. G., & Streuning, E. L. (1976). Life events, stress, and illness. Science, 194, 1013-1020.

Raskin, N. H., & Appenzeller, O. (1980). Headache. In L. H. Smith Jr. (Ed.), Major problems in internal medicine (vol. 19). Philadelphia: W.B. Saunders.

Rolf, L. H., Wiele, G., & Brune, G. G. (1981). 5-

Hydroxytryptamine in plateletes of patients with muscle-contraction headache. Headache, 21, 10-11.

Rowan, A. J. (1974). The electroencephalographic characteristics of migraine. Archives of Neurobiology, 37, 95.

Sacks, O. W. (1970). Migraine. Berkeley, CA: University of California press.

Sacks, O. W. (1981). Migraine. London: Pan Books.

Sakai, F., & Meyer, J. S. (1978). Regional cerebral haemodynamics during migraine and cluster headaches measured by the 133 xenon inhalation method. Headache, 18, 122-132.

Saper, J. (1978). Migraine II: Treatment. Journal of the American Medical Association, 239, 2480-2484.

Saper, J. R. (1983). The mixed headache syndrome: A new perspective. Headache, 23, 284-286.

Sarason, I. G., & Johnson, J. H. (1979). Life stress, organizational stress, and job satisfaction. Psychological Reports, 44, 75-79.

Sarason, I. G., Johnson, J. H., & Seigel, J. M. (1978). Assessing the impact of life changes: Development of the Life Experiences Survey. Journal of Consulting and Clinical Psychology, 40, 932-946.

Selby, G., & Lance, J. W. (1960). Observations on 500 cases of migraine and allied vascular headache. Journal of Neurology, Neurosurgery, and Psychiatry,

23, 230-232.

Sicuteri, F. (1959). Prophylactic and therapeutic properties of methyl lysurgic acid butanolamide in migraine. International Archives of Allergy, 15, 300.

Siegel, J. M., Johnson, J. H., & Sarason, I. G. (1979). Life changes and menstrual discomfort. Journal of Human Stress, 5, 41-46.

Skinhoj, E. (1973). Haemodynamic studies within the brain during migraine. Archives of Neurology, 29, 95-98.

Somerville, B. W. (1975). Estrogen-withdrawal migraine (1): Duration of exposure required and attempted prophylaxis by premenstrual estrogen administration. Neurology, 25, 239-244.

Somerville, B. W. (1972). The influence of progesterone and estradiol upon migraine. Headache, 12, 93-102.

Sovak, M., Kunzel, M., Sternbach, R., & Dalessio, D. (1981). Mechanism of the biofeedback therapy of migraine: Volitional manipulation of the psychophysiological background. Headache, 21, 89-92.

Steiner, T. J., Guha, P., Capildeo, R., & Rose, F. C. (1980). Migraine in patients attending a migraine clinic: An analysis by computer of age, sex, and family history. Headache, 20, 190-195.

Sternbach, R. A., Dalessio, D. J., Kunzel, M., & Bowman, G. E. (1980). MMPI patterns in common headach disorders. Headache, 20, 311-315.

Strauss, H., & Selinsky, H. (1941). EEG findings in patients with migrainous syndrome. Trans American Neurological Association, 67, 250.

Sturgis, E. T. (1981, November). Lability and reactivity in headache activity. Paper presented at the annual convention of the Association for the Advancement of Behavior Therapy, New York.

Tunis, M. M., & Wolff, H. G. (1954). Studies on headache: Cranial artery vasoconstriction and muscle contraction headache. Archives of Neurology and Psychiatry, 71, 425-434.

Tunis, M. M., & Wolff, H. G. (1953). Studies on headache: Long term observations of the reactivity of the cranial arteries in subjects with vascular headaches of the migraine type. Archives of Neurology and Psychiatry, 70, 551-557.

Tunis, M. M., & Wolff, H. G. (1952). Analysis of cranial artery pulse waves in patients with vascular headache of the migraine type. American Journal of Medical Science, 224, 565-568.

van Boxtel, A., & van der Ven, J. R. (1978). Differential EMG activity in subjects with muscle-contraction headaches related to mental effort. Headache, 18, 233-237.

Vaughn, R., Pall, M. L., & Haynes, S. N. (1977). Frontalis EMG responses to stress in subjects with frequent muscle-contraction headaches. Headache, 17, 313-317.

Vinokur, A., & Selzer, M. L. (1975). Desirable versus undesirable life events: Their relationship to stress and mental distress. Journal of Personality and Social Psychology, 32, 329-337.

Waters, W. E. (1971). Migraine: Intelligence, social class, and familial prevalence. British Medical Journal, 2, 77-78.

Waters, W. E., & O'Connor, P. J. (1975). Prevalence of migraine. Journal of Neurology, Neurosurgery, and Psychiatry, 38, 613-616.

Waters, W. E., & O'Connor, P. J. (1971). Epidemiology of headache and migraine in women. Journal of Neurology Neurosurgery, and Psychiatry, 54, 148-153.

Watson, D. (1982). Neurotic tendencies among chronic pain patients: An MMPI item analysis. Pain, 14, 365-385.

Weeks, R., Baskin, S., Rapoport, A., Sheftell, F., & Arrowsmith, F. (1983). A comparison of MMPI personality data and frontalis electromyographic readings in migraine and combination headache patients. Headache, 23, 75-82.

Whitehouse, D., Pappas, J. A., Escala, P. H., & Livingston,

S. (1967). Electroencephalographic changes in children with migraine. New England Journal of Medicine, 276, 33.

Williamson, D. A. (1981). Behavioral treatment of migraine and muscle contraction headaches: Outcome and Theoretical explanations. Progress in Behavior Modification, 2, 163-201.

Wolff, H. G. (1963). Headache and other head pain. New York: Oxford University Press.

Wolff, H. G. (1937). Personality factors and reactions of subjects with migraine. Archives of Neurology and Psychiatry, 37, 895.

Zeiss, A. M. (1980). Aversiveness versus change in the assessment of life stress. Journal of Psychosomatic Research, 24, 15-19.

Ziegler, D. K. (1979). Headache syndromes: Problems of definition. Psychosomatics, 20, 443-447.

Ziegler, D. K. (1978). Tension headache. Medical Clinics of North America, 62, 495-505.

Zuckerman, M. (1960). The development of an affect adjective checklist for the measurement of anxiety. Journal of Consulting Psychology, 24, 457-462.

Zuckerman, M., Albright, R. J., Marks, C. S., & Miller, G. L. (1961). Stress and hallucinatory effects of perceptual isolation and confinement. Psychological Monographs, 76, 30.

Zuckerman, M., & Lubin, B. (1965). Manual for the

Multiple Affect Adjective Check list. San Diego, CA: Educational and Industrial Testing Service.

Zuckerman, M., Lubin, B., & Robins, S. (1965). Validation of the Multiple Affect Adjective Check List in clinical situations. Journal of Consulting Psychology, 29, 594.

Zuckerman, M., Lubin, B., Vogel, L., & Valerius, E. (1964). Measurement of experimentally induced affects. Journal of Consulting Psychology, 28, 418-425.

Zuckerman, M., Nurnberger, J. I., Gardiner, S. H., Vandiveer, J. M., Barrett, B. H., & den Breeijen, A. (1962). Psychological correlates of somatic complaints in pregnancy and difficulty in childbirth. Journal of Consulting Psychology, 27, 324-329.

Appendix A

HEADACHE PROJECT
TELEPHONE SCREENING FORM

Name:_____ Sex: M F Age:_____

Phone:_____(home)_____(work)

How long have you been having headaches? _____

Have you seen a neurologist for this problem?_____When?_____

Neurological work up?_____What kind?_____

What diagnosis(es) were you given?_____

What medications are you currently taking for your HA?_____

_____

What other treatments have you sought for your HA's?_____

Description of head pain: (check all that apply)

___unilateral onset
___bilateral onset
___prodromes
___others:
_____
_____

___pulsating pain
___constant ache
___accompanied by nausea
___hypersensitivity to light
___hypersensitivity to noise

Frequency of headaches: _____more than three per week
_____2-3 per week
_____2-4 per month
_____less than 2 per month

TMJ?_____ Trauma at onset of problem?_____ Sinus Headaches____

Family history of headaches?_____

Other medical problems? _____

Two types of head pain? _____

Describe the headache project. Would you like to be scheduled for an intake interview?

Scheduled for:_____

Not scheduled because: ___needs neurological workup (CAT, xray, EEG)
___diagnosed TMJ ___trauma at onset of problem
___indistinguishable sinus headaches

DATE:_____ INTERVIEWER NAME:_____

Appendix B

CONSENT FOR DIAGNOSIS

I, _____, freely and willingly consent to participate in two interviews for the purpose of determining, if possible, a diagnosis for the type(s) of headaches I suffer. I understand that one interview will be conducted by a Board Certified Neurologist and one by a doctoral student in clinical psychology. Following these interviews, the neurologist and psychology student will discuss the information I have provided in order to: (1) attempt to reach an agreement concerning a diagnosis for my headaches; and (2) to decide whether or not my headaches are of the type they are examining their current research project.

In the event they determine that my headaches are appropriate for their project, I will have the choice of whether or not I wish to participate in the project. I understand that my decision at this time is not final and that I may decide not to participate, further, at any time I choose with no adverse consequences to me. In the event they determine that my headaches are not appropriate for their project, I will be given a full explanation of the reasons and will have suffered no adverse consequences with the exception of loosing time I spent in the one hour interview process.

Name _____ Date _____ Witness _____

Appendix H

INFORMED CONSENT

I, _____,
freely and willingly consent to be a participant in the research project investigating psychological, physiological, and environmental factors as they relate to headache activity. This project is being directed by Dr. Phillip J. Brantley of the L.S.U. Department of Psychology and of the L.S.U. Medical School. The project is being run and coordinated by several clinical psychology doctoral students from LSU.

I have been asked to attend biweekly assessment sessions at Earl K. Long Memorial Hospital which will last from approximately 1½ to 2 hours each. During these sessions I will be asked to fill out a number of paper and pencil questionnaires, to allow psychophysiological recordings to be taken, and to allow the drawing of blood samples. In addition to these biweekly sessions, I will also fill out several questionnaires at home on a daily basis. I understand that if I provide all the above information over a four week period, I will be able to receive treatment for my headaches at no cost to me.

I understand that the only risks involved are: (1) possible minor skin irritation due to the electrodes used in the psychophysiological assessments, (2) normal risks associated with any blood drawing procedure (conducted by R.N.) and (3) my usual headaches since no treatment will be offered during the four weeks of assessment.

I understand that this work is experimental in nature. I also understand that I may withdraw from participation in this study at any time with no adverse consequences. In addition, any information I provide during the project will be kept in strict confidence and if this information is presented publicly (i.e. conferences, journal articles), no information will be identified with me personally.

I realize that I have a right to ask questions at any time and to have these questions answered to my satisfaction. I have read and thoroughly understand this consent form.

_____ _____
Participant Witness

_____
Date

Appendix I

DEMOGRAPHIC INFORMATION

Subject number _____

Date of Birth: _____

Sex (circle): M F

Marital Status (circle):

1	2	3	4	5
single	married	divorced	widowed	separated

Number of persons living in your household (include yourself): ____

Education completed (check one):

1. _____did not finish high school
2. _____graduated high school (or have equivalent, e.g., GED)
3. _____attended college but have no degree
4. _____have a 2 year college degree (or equivalent)
5. _____have a 4 year college degree or more

Annual income level of household (check one):

1. _____less than $10,000
2. _____$10,000 - $29,000
3. _____$30,000 - $49,000
4. _____$50,000 - $99,000
5. _____$100,000 +

Appendix J

Analysis of Variance Summary Table for Age Differences Between Diagnostic Groups

Source	df	SS	MS	F	p
Diagnosis	3	473.65	157.88	1.53	NS
Error	76	7856.30	103.37		
Total	79	8329.95			

Appendix K

Analysis of Variance Summary Tables for Group Differences in Headache Activity

Source	df	SS	MS	F	p
		Headache Frequency			
Diagnosis	3	3695.44	1231.81	29.31	.0001
Error	76	3193.95	42.03		
Total	79	6889.39			
		Headache Intensity			
Diagnosis	3	30.83	10.38	25.04	.0001
Error	76	31.19	.41		
Total	79	62.02			
		Headache Duration			
Diagnosis	3	455.98	151.99	14.08	.0001
Error	76	820.68	10.80		
Total	79	1276.66			

Appendix L

Summary Tables for Multiple Regression Analyses Predicting Headache Frequency

Source	df	SS	MS	F	p	R^2
Model	5	596.10	119.22	2.35	.053	.178
Diagnosis	2	461.43[a]	230.71	4.54	.015	
MAACL-ANX	1	8.42[a]	8.42	.17	.685	
Interaction	2	126.25[a]	63.13	1.24	.297	
Error	54	2744.08	50.82			
Total	59	3340.18				
Model	5	654.36	130.87	2.63	.034	.196
Diagnosis	2	461.43	230.71	4.64	.014	
MAACL-DEP	1	2.27	2.27	.05	.832	
Interaction	2	190.66	95.33	1.92	.157	
Error	54	2685.82	49.74			
Total	59	3340.18				
Model	5	676.56	135.31	2.74	.028	.203
Diagnosis	2	461.43	230.71	4.68	.013	
MAACL-HOS	1	123.84	123.84	2.51	.119	
Interaction	2	91.30	45.65	.93	.403	
Error	54	2663.62	49.33			
Total	59	3340.18				

Appendix L (con't)

Model	5	557.47	111.49	2.16	.072	.167
Diagnosis	2	461.43	230.71	4.48	.016	
DSI-FREQ	1	40.10	40.10	.78	.382	
Interaction	2	55.93	27.97	.54	.584	
Error	54	2782.71	51.53			
Total	59	3340.18				

Model	5	521.90	104.38	2.00	.093	.156
Diagnosis	2	461.43	230.71	4.42	.017	
DSI-SUM	1	13.48	13.48	.26	.613	
Interaction	2	46.99	23.49	.45	.640	
Error	54	2818.28	52.19			
Total	59	3340.18				

Model	5	502.34	100.47	1.91	.107	.150
Diagnosis	2	461.43	230.71	4.39	.017	
NEG NUM FIRST	1	.02	.02	.00	.986	
Interaction	2	40.89	20.45	.39	.680	
Error	54	2837.85	52.55			
Total	59	3340.18				

Appendix L (con't)

Model	5	486.37	97.27	1.84	.120	.146
Diagnosis	2	461.43	230.71	4.37	.017	
NEG NUM LAST	1	3.30	3.30	.06	.803	
Interaction	2	21.63	10.81	.20	.815	
Error	54	2853.81	52.85			
Total	59	3340.18				

Model	5	529.41	105.88	2.03	.088	.158
Diagnosis	2	461.43	230.71	4.43	.017	
TOTAL NEG NUM	1	.78	.78	.02	.903	
Interaction	2	67.19	33.59	.65	.528	
Error	54	2810.77	52.05			
Total	59	3340.18				

Model	5	483.92	96.78	1.83	.123	.145
Diagnosis	2	461.43	230.71	4.36	.017	
NEG SUM FIRST	1	.01	.01	.00	.989	
Interaction	2	22.48	11.24	.21	.809	
Error	54	2856.26	52.89			
Total	59	3340.18				

Appendix L (con't)

Model	5	482.58	96.52	1.82	.124	.144
Diagnosis	2	461.43	230.71	4.36	.018	
NEG SUM LAST	1	8.18	8.18	.15	.696	
Interaction	2	12.96	6.48	.12	.885	
Error	54	2857.60	52.92			
Total	59	3340.18				
Model	5	488.30	97.66	1.85	.119	.146
Diagnosis	2	461.43	230.71	4.37	.017	
TOTAL NEG SUM	1	1.97	1.97	.04	.848	
Interaction	2	24.90	12.45	.24	.791	
Error	54	2851.88	52.81			
Total	59	3340.18				
Model	5	654.85	130.97	2.63	.033	.196
Diagnosis	2	461.43	230.71	4.64	.014	
MAACL-INT	1	30.18	30.18	.61	.439	
Interaction	2	163.24	81.62	1.64	.203	
Error	54	2685.33	49.73			
Total	59	3340.18				

Note. Diagnosis was entered in all regressions, whereas each stress and affect variable was entered only in its own equation.

*Type 1 Sums of Squares.

Appendix M

Summary Tables for Multiple Regression Analyses Predicting Headache Intensity

Source	df	SS	MS	F	p	R^2
Model	5	.50	.10	.28	.923	.025
Diagnosis	2	.47[a]	.23	.67	.518	
MAACL-ANX	1	.00[a]	.00	.01	.920	
Interaction	2	.02[a]	.01	.02	.976	
Error	54	19.25	.36			
Total	59	19.75				
Model	5	.77	.15	.44	.818	.039
Diagnosis	2	.47	.23	.68	.513	
MAACL-DEP	1	.01	.01	.02	.898	
Interaction	2	.29	.15	.42	.661	
Error	54	18.97	.35			
Total	59	19.75				
Model	5	.78	.16	.45	.815	.040
Diagnosis	2	.47	.23	.68	.513	
MAACL-HOS	1	.15	.15	.42	.520	
Interaction	2	.16	.08	.23	.796	
Error	54	18.96	.35			
Total	59	19.75				

Appendix M (con't)

Model	5	1.58	.32	.94	.462	.080
Diagnosis	2	.47	.23	.71	.498	
DSI-FREQ	1	.41	.41	1.23	.272	
Interaction	2	.69	.35	1.03	.364	
Error	54	18.16	.34			
Total	59	19.75				
Model	5	1.41	.28	.83	.535	.071
Diagnosis	2	.47	.23	.70	.502	
DSI-SUM	1	.04	.04	.12	.732	
Interaction	2	.89	.45	1.31	.277	
Error	54	18.34	.34			
Total	59	19.75				
Model	5	.93	.19	.53	.749	.047
Diagnosis	2	.47	.23	.68	.510	
NEG NUM FIRST	1	.24	.24	.70	.406	
Interaction	2	.21	.10	.31	.738	
Error	54	18.81	.35			
Total	59	19.75				

Appendix M (con't)

Model	5	1.03	.21	.60	.703	.052
Diagnosis	2	.47	.23	.68	.508	
NEG NUM LAST	1	.04	.04	.11	.746	
Interaction	2	.52	.26	.75	.477	
Error	54	18.71	.35			
Total	59	19.75				
Model	5	.65	.13	.37	.869	.033
Diagnosis	2	.47	.23	.67	.515	
TOTAL NEG NUM	1	.10	.10	.28	.600	
Interaction	2	.07	.03	.11	.900	
Error	54	19.10	.35			
Total	59	19.75				
Model	5	.74	.15	.42	.831	.038
Diagnosis	2	.47	.23	.67	.514	
NEG SUM FIRST	1	.07	.07	.19	.663	
Interaction	2	.20	.10	.29	.752	
Error	54	19.00	.35			
Total	59	19.75				

Appendix M (con't)

Model	5	1.21	.24	.71	.522	.061
Diagnosis	2	.47	.23	.69	.505	
NEG SUM LAST	1	.31	.31	.89	.349	
Interaction	2	.43	.26	.63	.538	
Error	54	18.54	.34			
Total	59	19.75				
Model	5	.52	.10	.29	.916	.026
Diagnosis	2	.47	.23	.67	.518	
TOTAL NEG SUM	1	.00	.00	.00	.969	
Interaction	2	.04	.02	.06	.944	
Error	54	19.23	.36			
Total	59	19.75				
Model	5	.62	.12	.35	.880	.031
Diagnosis	2	.47	.23	.67	.516	
MAACL-INT	1	.03	.03	.10	.759	
Interaction	2	.11	.06	.16	.854	
Error	54	19.13	.35			
Total	59	19.75				

Note. Diagnosis was entered in all regressions, whereas each stress and affect variable was entered only in its own equation.

*Type 1 Sums of Squares.

Appendix N

Summary Tables for Multiple Regression Analyses Predicting Headache Duration

Source	df	SS	MS	F	p	R^2
Model	5	29.96	5.99	.47	.799	.041
Diagnosis	2	15.60[a]	7.80	.61	.548	
MAACL-ANX	1	1.93[a]	1.93	.15	.700	
Interaction	2	12.43[a]	6.27	.48	.619	
Error	54	692.40	12.82			
Total	59	722.36				
Model	5	34.64	6.93	.54	.742	.048
Diagnosis	2	15.60	7.80	.61	.546	
MAACL-DEP	1	6.61	6.61	.52	.474	
Interaction	2	12.43	6.27	.49	.617	
Error	54	687.72	12.74			
Total	59	722.36				
Model	5	18.89	3.78	.29	.917	.026
Diagnosis	2	15.60	7.80	.60	.553	
MAACL-HOS	1	.03	.03	.00	.961	
Interaction	2	3.25	1.62	.12	.883	
Error	54	703.47	13.03			
Total	59	722.36				

Appendix N (con't)

Model	5	55.90	11.18	.91	.484	.077
Diagnosis	2	15.60	7.80	.63	.535	
DSI-FREQ	1	4.40	4.40	.36	.553	
Interaction	2	35.90	17.95	1.45	.243	
Error	54	18.16	.34			
Total	59	722.36				
Model	5	65.88	13.18	1.08	.380	.091
Diagnosis	2	15.60	7.80	.64	.530	
DSI-SUM	1	2.54	2.54	.21	.649	
Interaction	2	47.73	23.87	1.96	.150	
Error	54	656.48	12.16			
Total	59	722.36				
Model	5	37.24	7.45	.59	.710	.052
Diagnosis	2	15.60	7.80	.61	.545	
NEG NUM FIRST	1	15.63	15.63	1.23	.272	
Interaction	2	6.02	3.01	.24	.790	
Error	54	685.11	12.69			
Total	59	722.36				

Appendix N (con't)

Model	5	101.40	20.28	1.76	.136	.140
Diagnosis	2	15.60	7.80	.68	.512	
NEG NUM LAST	1	1.28	1.28	.11	.740	
Interaction	2	84.52	42.26	3.67	.032	
Error	54	620.96	11.50			
Total	59	722.36				
Model	5	54.09	10.82	.87	.505	.075
Diagnosis	2	15.60	7.80	.63	.536	
TOTAL NEG NUM	1	7.46	7.46	.60	.441	
Interaction	2	31.03	15.51	1.25	.294	
Error	54	668.27	12.38			
Total	59	722.36				
Model	5	30.01	6.00	.47	.798	.041
Diagnosis	2	15.60	7.80	.61	.548	
NEG SUM FIRST	1	10.90	10.90	.85	.361	
Interaction	2	3.51	1.75	.14	.873	
Error	54	692.35	12.82			
Total	59	722.36				

Appendix N (con't)

Model	5	146.47	29.29	2.75	.028	.203
Diagnosis	2	15.60	7.80	.73	.486	
NEG SUM LAST	1	4.68	4.68	.44	.511	
Interaction	2	126.19	63.10	5.92	.005	
Error	54	575.88	10.66			
Total	59	722.36				
Model	5	38.94	7.79	.62	.589	.054
Diagnosis	2	15.60	7.80	.62	.544	
TOTAL NEG SUM	1	3.75	3.75	.30	.589	
Interaction	2	19.59	9.79	.77	.466	
Error	54	683.42	12.66			
Total	59	722.36				
Model	5	27.37	5.47	.43	.829	.038
Diagnosis	2	15.60	7.80	.61	.549	
MAACL-INT	1	1.84	1.84	.14	.707	
Interaction	2	9.92	4.96	.39	.682	
Error	54	694.99	12.87			
Total	59	722.36				

Note. Diagnosis was entered in all regressions, whereas each stress and affect variable was entered only in its own equation.

*Type 1 Sums of Squares.

Appendix O

Summary Tables for Hierarchical Stepwise Multiple Regressions Predicting Headache Frequency From Measures of Major and Minor Stressful Life-events

Source	df	SS	MS	F	p	R^2
(a) DSI Scores Entered First[a]						
Regression	2	551.48	275.74	3.35	.040	.081
Error	77	6337.90	82.31			
Total	79	6889.39				

	B Value	STD Error	SS[b]	F	p
Intercept	16.78				
DSI-FREQ	-.67	.30	426.10	5.18	.026
DSI-SUM	.13	.11	130.38	1.58	.212

Source	df	SS	MS	F	p	R^2
(b) LES Scores Entered First[c]						
Regression	3	431.28	143.76	1.69	.174	.063
Error	76	6458.11	84.98			
Total	79	6889.39				

Appendix O (con't)

	B Value	STD Error	SS[b]	F	p
Intercept	16.69				
TOTAL NEG NUM	-.10	1.19	.60	.01	.933
TOTAL NEG SUM	.10	.55	3.02	.04	.851
DSI-FREQ[d]	-.36	.16	410.02	4.83	.031

Note. N = 80.

[a]Scores from the Daily Stress Inventory were entered and other variables were allowed to enter only at $p < .10$ level of significance.

[b]Type II Sums of Squares.

[c]Scores from the Life Experiences Survey were entered and other variables were allowed to enter only at $p < .10$ level of significance.

[d]Entered significantly at second step.

Appendix P

Summary Tables for Hierarchical Stepwise Multiple Regressions Predicting Headache Intensity From Measures of Major and Minor Stressful Life-events

Source	df	SS	MS	F	p	R^2
(a) DSI Scores Entered First[a]						
Regression	2	7.75	3.87	5.50	.006	.125
Error	77	54.27	.70			
Total	79	62.01				

	B Value	STD Error	SS[b]	F	p
Intercept	1.99				
DSI-FREQ	-.09	.03	7.73	10.97	.001
DSI-SUM	.03	.01	5.20	7.37	.008

Source	df	SS	MS	F	p	R^2
(b) LES Scores Entered First[c]						
Regression	4	8.27	2.07	2.89	.028	.133
Error	75	53.74	.72			
Total	79	62.01				

Appendix P (con't)

	B Value	STD Error	SS[b]	F	p
Intercept	2.04				
TOTAL NEG NUM	-.08	.11	.42	.59	.446
TOTAL NEG SUM	.03	.05	.25	.36	.553
DSI-FREQ[d]	-.09	.03	7.53	10.25	.002
DSI-SUM[d]	.03	.01	5.17	7.21	.009

Note. N = 80.

[a]Scores from the Daily Stress Inventory were entered and other variables were allowed to enter only at p < .10 level of significance.

[b]Type II Sums of Squares.

[c]Scores from the Life Experiences Survey were entered and other variables were allowed to enter only at p < .10 level of significance.

[d]Entered significantly at second step.

Appendix Q

Summary Tables for Hierarchical Stepwise Multiple Regressions Predicting Headache Duration From Measures of Major and Minor Stressful Life-events

Source	df	SS	MS	F	p	R^2
(a) DSI Scores Entered First[a]						
Regression	2	134.55	67.27	4.54	.014	.105
Error	77	1142.12	14.83			
Total	79	1276.66				

	B Value	STD Error	SS[b]	F	p
Intercept	6.62				
DSI-FREQ	-.38	.13	134.37	9.06	.003
DSI-SUM	.12	.04	100.20	6.76	.011

Source	df	SS	MS	F	p	R^2
(b) LES Scores Entered First[c]						
Regression	2	15.00	7.50	.46	.634	.012
Error	77	1261.66	16.39			
Total	79	1276.66				

Appendix Q (con't)

	B Value	STD Error	SS[b]	F	p
Intercept	6.07				
TOTAL NEG NUM	-.47	.52	13.47	.82	.367
TOTAL NEG SUM	.18	.24	9.36	.57	.452

Note. N = 80.

[a]Scores from the Daily Stress Inventory were entered and other variables were allowed to enter only at $p < .10$ level of significance.

[b]Type II Sums of Squares.

[c]Scores from the Life Experiences Survey were entered and other variables were allowed to enter only at $p < .10$ level of significance.

Appendix R

Analysis of Variance Summary Tables for Group Differences on Stress and Affect Measures Across Headache and Nonheadache Days

Source	df	SS[a]	F	p	η^2
		MAACL-Anxiety[d]			
Diagnosis[b]	2	14.53	.46	.635	.011
Error	56	887.57			
Day[c]	2	54.60	7.20	.001	.042
Interaction	4	13.55	.89	.471	.010
Error	89				
		MAACL-Depression[d]			
Diagnosis	2	52.59	.83	.442	.019
Error	56	1779.35			
Day	2	159.81	10.21	.000	.057
Interaction	4	129.13	4.13	.004	.046
Error	89	695.27			

Appendix R (con't)

		MAACL-Hostility[d]			
Diagnosis	2	26.08	.97	.386	.020
Error	56	753.25			
Day	2	30.87	3.31	.041	.024
Interaction	4	83.42	4.48	.002	.064
Error	89	414.71			
		DSI-FREQ*			
Diagnosis	2	220.84	1.32	.276	.038
Error	56	4689.02			
Day	2	74.39	3.96	.023	.013
Interaction	4	22.83	.61	.658	.004
Error	89	835.14			
		DSI-SUM*			
Diagnosis	2	2400.49	1.43	.248	.042
Error	56	47045.42			
Day	2	810.67	4.98	.009	.014
Interaction	4	64.30	.20	.939	.001
Error	89	7243.80			

Appendix R (con't)

		MAACL-Intensity[f]			
Diagnosis	2	1186.65	.77	.468	.017
Error	56	43193.99			
Day	2	3075.95	7.32	.001	.045
Interaction	4	2740.23	3.26	.015	.040
Error	89	18710.45			

Note. These are post-hoc analyses from an overall significant MANOVA.

[a]Type III Sums of Squares.

[b]Migraine, mixed, and muscle-contraction headache groups.

[c]Includes separate scores on the dependent measures from 2 days prior to a headache, 1 day prior to a headache, and headache days.

[d]Scores from the Multiple Affect Adjective Checklist averaged over headache and nonheadache days.

[e]Scores from the Daily Stress Inventory averaged over headache and nonheadache days.

[f]Sum of the three MAACL scores after transformed to t scores.

Appendix S

Post-hoc Scheffe Comparisons for Significant Interactions Between Headache Groups Across Headache and Nonheadache Days on MAACL Depression Scores

	Group Comparison	
Days	M-C vs. Migraine	M-C vs. Mixed
1 vs. 3[a]	.34	1.90
1 vs. 2[b]	4.37*	4.43*

[a]Scores one day prior to a headache versus headache day scores.

[b]Scores one day prior to a headache versus scores two days prior to a headache.

*$p < .05$

Appendix T

Post-hoc Scheffe Comparisons for Significant Interactions Between Headache Groups Across Headache and Nonheadache Days on MAACL Hostility Scores

	Group Comparison	
Days	M-C vs. Migraine	M-C vs. Mixed
1 vs. 3[a]	-.44	.73
1 vs. 2[b]	3.88*	3.59*

[a]Scores one day prior to a headache versus headache day scores.

[b]Scores one day prior to a headache versus scores two days prior to a headache.

*$p < .05$

Appendix U

Post-hoc Scheffe Comparisons for Significant Interactions Between Headache Groups Across Headache and Nonheadache Days on MAACL Total Negative Affect Scores

	Group Comparison	
Days	M-C vs. Migraine	M-C vs. Mixed
1 vs. 3[a]	1.55	6.84
1 vs. 2[b]	22.60	20.49

[a]Scores one day prior to a headache versus headache day scores.

[b]Scores one day prior to a headache versus scores two days prior to a headache.

CURRICULUM VITA

CRAIG DOUGLAS WAGGONER

Home Address:
1574-P Bridle Circle
Greenville, NC 27834
(919) 756-9431

Work Address:
Pitt County Memorial
Hospital Regional
Rehabilitation Center
Department of Psychology
P.O. Box 6028
Greenville, NC 27834
(919) 757-4318

EDUCATIONAL BACKGROUND:

1981 - 1986	Louisiana State University Baton Rouge, Louisiana Doctoral Candidate Major Area: Clinical Psychology Specialty Area: Behavioral Medicine Minor Area: Behavioral Neurology Degree: Ph.D., May, 1986
1978 - 1980	Bradley University Peoria, Illinois Major Area: Clinical/Community Psychology Degree: M.A., May, 1980
1974 - 1978	Ohio State University Columbus, Ohio Major Area: Psychology Degree: B.S., June, 1978

CLINICAL EXPERIENCE:

8/85 - Present	Staff Psychologist Pitt County Memorial Hospital-Regional Rehabilitation Center, Department of Psychology, Greenville, NC Conduct psychological assessments and provide treatment to inpatients with spinal cord injuries or CVAs as part of multidisciplinary treatment team. Supervisor: Gregory L. Duncan, Ph.D.
9/84 - 8/85	Clinical Psychology Intern (as doctoral candidate): UMDNJ-Rutgers Medical School, Dept. of Psychiatry

Conducted psychological assessments and provided treatment for outpatients. Full-time experience divided primarily among Middlesex General-University Hospital (2/3 time), the Behavior Therapy Clinic (1/6 time), and the Sexual Counseling Clinic (1/6 time).
Supervisors: Lynn Pendarvis Clemow, Ph.D.
Paul Lehrer, Ph.D.
Raymond Rosen, Ph.D.

9/82 - 8/84 — Medical Psychology Trainee:
Earl K. Long Memorial Hospital, Family Practice Unit, Baton Rouge, LA
Conducted psychological assessments and treatment for child, adolescent, and adult outpatients referred from family practice physicians (20 hrs./wk).
Supervisor: Phillip J. Brantley, Ph.D.

6/82 - 8/82 — Adult Psychology Trainee:
Psychological Services Center, Louisiana State University, Baton Rouge, LA
Practicum experience in conducting psychological assessments and treatment of adult disorders (10 hrs./wk).
Supervisor: William F. Waters, Ph.D.

1/82 - 5/82 — Child Psychology Trainee:
The Developmental Center, Louisiana State University
Practicum experience in conducting psychological assessments and treatment of disorders of childhood.
(5 hrs./wk).
Supervisor: Ralph M. Dreger, Ph.D.

12/79 - 7/81 — Clinical Therapist:
Human Service Center, Peoria, IL
Full-time paid position (Master's level)
Conducted psychological assessments and treatment with post-hospitalized adults in individual and group modalities.
Psychological testing (WAIS, MMPI, Halstead-Reitan Battery).
Supervisor: David H. Gilliland, Psy.D.

8/80 - 6/81 — Neuropsychology Trainee:
Neuropsychology Center, Peoria, IL
Practicum experience in administration, scoring, and interpretation of the Halstead-Reitan Neuro-psychological Battery (3 hrs/wk).

Supervisor: Robert B. LeLieuvre, Ph.D.

8/79 - 12/79 — Clinical Psychology Intern (as master's candidate):
Human Service Center, Peoria, IL
Responsibilities the same as Clinical Therapist (above) with additional supervision (36 hrs./wk).
Supervisor: David H. Gilliland, Psy.D.

CLINICAL SUPERVISORY EXPERIENCE:

8/85 - Present — Psychological Associate in Department of Psychology of Pitt County Memorial Hospital, Greenville, NC
Provide clinical training to Master's level interns (from East Carolina University) during their assessments and treatment of inpatients at the Regional Rehabilitation Center.
Supervisor: Gregory L. Duncan, Ph.D.

9/84 - 8/85 — Clinical Psychology Intern in the Departmentof Psychiatry, Middlesex General University Hospital,
New Brunswick, NJ
Supervisor to graduate students (attending Rutgers University's doctoral program in clinical psychology) during theirassess-ments and treatments of outpatient referrals to the Center for Stress Manage-ment and Behavioral Medicine.
Supervisor: Lynn Pendarvis Clemow, Ph.D.

8/83 - 8/84 — Chief Extern and Coordinator of Psychology Service in Family Practice Unit, Earl K. Long Memorial Hospital, Baton Rouge, LA
Supervisor to three clinical psychology graduate students from Louisiana State University. Coordinated all patient referrals and monitored their dispositions in psychology services (1/2 time paid position).
Supervisor: Phillip J. Brantley, Ph.D.

CONSULTATION EXPERIENCE:

8/85 - Present — Pitt County Memorial Hospital, Greenville, NC
Consult to physicians and nursing staff treating medical inpatients on acute wards.
Supervisor: Gregory L. Duncan, Ph.D.

11/83 - 8/84	Baton Rouge Renal Center, Baton Rouge, LA Conducted Stress Management program for the nursing staff. Conducted individual assessments and treatments with patients receiving regular intermittent hemodialysis. Supervisor: Phillip J. Brantley, Ph.D.
6/83 - 8/84	Consultation and Liaison Service, Department of Medicine, Earl K. Long Memorial Hospital Provided inpatientassessments/recommendations to health care staff. Provided emergency room consultations on "on-call" basis. Supervisor: Phillip J. Brantley, Ph.D.
9/82 - 8/83	Employee Assistance Program and Talbot Outpatient Alcohol and Drug Treatment Center, Baton Rouge, LA Provided EAP services to companies on contract in Baton Rouge area. Provided assessments and treatment to outpatients referred from Talbot Center. Developed and conducted assertiveness training program. Supervisor: Donald A. Williamson, Ph.D.
3/82 - 6/82	Pain Treatment Center of Baton Rouge General Hospital, Baton Rouge, LA Provided assessments and treatment to chronic pain inpatients. Co-conducted chronic pain group. Supervisor: Phillip J. Brantley, Ph.D.

TEACHING EXPERIENCE:

1981 - 1982 (academic year)	Graduate Teaching Assistant: Department of Psychology, Louisiana State University Supervisor: Donald A. Williamson, Ph.D.
1978 - 1979 (academic year)	Graduate Teaching Assistant: Department of Psychology, Bradley University Supervisors: Robert H. Lowder, Ph.D. Lawrence C. Walker, Ph.D.

AWARDS/HONORS:

1981 - 1982	National Institute of Mental Health Fellow
1980	Illinois Psychological Association Graduate Research Award

1979 — Bradley University Research and Creativity Award

GRANTS:

1980 — (Co-author). A method to increase compliance to exercise regimens in rheumatoid arthritis patients. Sponsor: Bradley University ($1,076). Completed.

RESEARCH EXPERIENCE:

10/84 - 8/85 — Coordinated research conducted atMiddlesex General University Hospital investigating psychological, psychophysiological, and behavioral parameters of patients presenting with angina-like chest pains. Research Director: Lynn P. Clemow, Ph.D.

8/82 - 8/84 — Coordinated research conducted at LSU and Earl K. Long Memorial Hospital investigating the measurement of stress, stress and illness relationships, and psychological and psychopsychological parameters of chronic headache patients.
Research Director: Phillip J. Brantley, Ph.D.

1981 - 1982 — Member of research team investigating behavioral cognitive, andpsychophysiological correlates of heterosocially anxious males. LSU Department of Psychology.

PUBLICATIONS:

Brantley, P.J., Waggoner, C.D., Jones, G.N., & Rappaport, N.B. (in press). A daily stress inventory: Development, reliability, and validity. Journal of Behavioral Medicine.

Faustich, M., Gresham, F., McAnulty, D., Veitia, M., Moore, J., Bernard, B., Waggoner, C., & Howell, R. (in press). Factor structure of the WAIS-R for an incarcerated population. Journal of Clinical Psychology.

Rappaport, N., McAnulty, D., Waggoner, C., & Brantley, P., (in press). Cluster analysis of MMPI profiles in a headache population. Journal of Behavioral Medicine.

Rappaport, N., McAnulty, P., Waggoner, C., Brantley, P., Barkemeyer, C., & McKenzie, S. (1986). Psychopathology in volunteers for headache research. Initial vs. later respondents. Headache, 26, 37-38.

Waggoner, C.D. & LeLieuvre, R.B. (1984). A method to increase compliance to exercise regimens in rheumatoid arthritis patients. Journal of Behavioral Medicine, 4(2), 191-201.

Williamson, D.A. & Waggoner, C.D. Behavioral and pharmacological treatment of psychophysiological disorders (1986). In M. Hersen, Pharmalogical and behavioral treatment: An integrative approach.

MANUSCRIPTS SUBMITTED OR IN PREPARATION;

Callon, E., Brantley, P., McAnulty, D., Waggoner, C., & Rappaport, N. (submitted). The effects of headache chronicity on frontal EMG of muscle-contraction headache sufferers.

McAnulty, D., Graham, F., Faustich, M., Veitia, M. Howell, R., Waggoner, C., Moore, J., & Bernard (submitted). Two- and four-subtest WAIS-R short forms for an incarcerated population.

McAnulty, D., Rappaport, N., Waggoner, C., & Brantley, P. (submitted). Psychological correlates of head pain.

Waggoner, C., McAnulty, P., Rappaport, N., Goreczny, A., & Brantley, P. (in preparation). Headache activity, chronicity, and illness behavior: A multivariate analysis.

Waggoner, C., Brantley, P., Rappaport, N., McAnulty, P., & Goreczny, A. (in preparation). Evidence for a stress-headache relationship: The importance of daily stress.

Waggoner, C., Brantley, P., McAnulty, P., Rappaport, N., & Goreczny, A. (in preparation). Patterns of daily mood changes in relation to migraine, muscle-contraction, and combination headache attacks.

CURRENT RESEARCH INTERESTS:

Measurement of "stress" and its relationship to psychological and physical disorders.

Psychological, behavioral, and psychophysiological parameters of headache.

Assessment and treatment of pain and illness behavior in SCI patients.

Psychophysiological assessment and treatment of involuntary muscle-spasms in SCI patients.

PAPER PRESENTATIONS:

Bruce, B., Waggoner, C. & Williamson, D.(1983) Evaluation of response generalization in the treatment of agoraphobia. Presented at the annual meeting of the Southeastern Psychological Association, Atlanta, GA.

Jensen, B., Williamson, D., Waggoner, C., Turin, C., & Lozada, S. (1986). Psychophysiological and observational assessment of heterosocial competence: A methodological investigation. Presented at the annual meeting of the Southeastern Psychological Association, Orlando, FL.

Jones, G., Gilchrist, J., Goreczny, A., Waggoner, C., Ruggiero, L., & Valinchus, T. (1986). The daily stress inventory: Validity and the effect of repeated administration. Presented at the annual meeting of the Southeastern Psychological Association, Orlando, FL.

McAnulty, D, Rappaport, N., Waggoner, C., Barkemeyer, C., & Himel, T. (1984). Reliability of symptom reports in headache: Implications for the validity of diagnosis. Presented at the annual meeting of the SoutheasternPsychological Association, New Orleans, LA.

McAnulty, D., Rappaport, N., Waggoner, C., & Brantley, P. (1984). Comprehensive assessment of migraine: A single case study. Presented at the annual meeting of the Southeastern Psychological Association, New Orleans, LA.

McAnulty, D., Rappaport, N., Waggoner, C. & Brantley, P. (1984). Psychological correlates of head pain. Presented at the annual meeting of ABBT.

McAnulty, D., Rappaport, N., Waggoner, C., McKenzie, S., & Brantley, P. (1985). The relationship between depression and headache parameters. Presented at the annual meeting of the Southeastern Psychological Association, Atlanta, GA.

Rappaport, N., McAnulty, D., Waggoner, C., McKenzie, S., & Brantley, P. (1984). Cluster analysis of MMPI profile in a headache population. Presented at the annual meeting of the Southeastern Psychological Association, New Orleans, LA.

Rappaport, N., McAnulty, D., Waggoner, C., McKenzie, S., & Brantley, P. (1985). Psychopathology in volunteers for headache research: Initial vs. later respondents. Presented at the annual meeting of the Southeastern Psychological Association, Atlanta, GA.

Waggoner, C., Jones, G. & Brantley, P. (1984). The distribution of daily stress among sociodemographic categories. Presented at the 1984 annual meeting of AABT, Philadelphia, PA.

Waggoner, C, & LeLieuvre, R. (1980). A method to increase compliance to exercise regimens in rheumatoid arthritis patients. Presented at annual meeting of the Midwestern Psychological Association, St. Louis, MO.

Waggoner, C., McAnulty, D., Rappaport, N., Goreczny, A., Jones, G. & Brantley, P. (1984). The relationship between headache activity, chronicity, and illness behavior. Presented at the annual meeting of AABT, Philadelphia, PA.

Waggoner, C., McAnulty, D., Rappaport, N. & Ruggiero, L. (1983). An investigation of four correlates of migraine headache. Presented at the combined meeting of AABT and the World Congress on Behavior Therapy, Washington, DC.

Waggoner, C., Rappaport, N., Jones, & Brantley, P. (1983). A daily stress record: Development, reliability, and validity. Presented at the combined meeting of AABT and the World Congress on Behavior Therapy, Washington, DC.

Williamson, D., Jensen, B., Waggoner, C., Turin, C. & Lozada, S. (1984). Psychophysiological assessment of heterosocial anxiety. Presented at the annual meeting of AABT, Philadelphia, PA.

Williamson, D., Labbe, E., Schellinger, J., & Waggoner, C. (1982). Behavioral treatment of headache: Psychophysiological bases of therapeutic benefits. Presented at the annual meeting of ABBT, Los Angeles, CA.

REFERENCES;

Phillip J. Brantley, Ph.D.
Associate Professor of Psychology and Family Medicine
Department of Psychology
Louisiana State University
Baton Rouge, LA 70803

Lynn P. Clemow, Ph.D.
UMDNJ-Rutgers Medical School
Department of Psychiatry
Middlesex General-University Hospital
180 Somerset Street
New Brunswick, NJ 08903

William F. Waters, Ph.D.
Director of Clinical Training
Professor, Department of Psychology
Louisiana State University
Baton Rouge, LA 70803

Gregory L. Duncan, Ph.D.
Chief of Psychology
Pitt County Memorial Hospital
Regional Rehabilitation Center
P.O. Box 6028
Greenville, NC 27834

DOCTORAL EXAMINATION AND DISSERTATION REPORT

Candidate: Craig Douglas Waggoner

Major Field: Psychology

Title of Dissertation: The Relation Among Stressful Life-events, Affective Responses, and Headaches

Approved:

Major Professor and Chairman

Dean of the Graduate School

EXAMINING COMMITTEE:

Date of Examination:

April 21, 1986